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# CEREBRAL MANIFESTATIONS FOLLOWING ACUTE OTITIS MEDIA IN INFANCY AND IN EARLY CHILDHOOD

## WITH PARTICULAR REFERENCE TO THE OCCURRENCE OF JACKSONIAN CONVULSIONS, CONJUGATE DEVIATION OF THE HEAD AND EYES AND HEMIPLEGIA

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In the course of acute infectious diseases of infancy and early childhood, certain cerebral manifestations, especially those indicating involvement of the motor cortex, are not uncommonly observed. These symptoms have been ascribed to a toxic or a general inflammatory reaction of the cerebral cortex or to a vascular lesion consequent to changes in the blood vessels. The condition has come to be known as acute nonsuppurative encephalitis. The term, unfortunately, does not indicate a specific clinical entity and is not associated with a clearcut pathologic picture. It has been applied to any presumed cerebral lesion in patients presenting the history of acute onset of an infectious state which, early in its course, was accompanied by cerebral symptoms. The most common manifestations in such instances are generalized or localized convulsions, which may or may not be followed by hemiplegia. In some instances, the predisposing infection is known, such as one of the common communicable diseases or an infection of the respiratory passages. A large group of cases exists, however, in which the focus or the nature of the infection has not been determined, and for this reason the cerebral lesion has been designated as primary. It is to this group that we wish to call attention.

As neurologists collaborating with physicians in the services of communicable diseases, pediatrics and otology of the Los Angeles County Hospital, we have had the opportunity to study a group of children who presented pronounced unilateral motor manifestations, associated with either an obvious infection of the middle ear or one accompanied by few or no clinical symptoms or signs. At times otitis media was evidently a direct consequence of an acute infectious disease, but in other instances it was apparently primary. It was perhaps the dramatic

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outcome of the first case which came under our observation which focused our attention particularly on the infection in the middle ear, rather than on the acute general infection, as the probable cause of the cerebral symptoms and of the predominant localization of the trouble in the parietal lobe. The similarity in clinical aspects, as presented by a group of 23 patients seems to indicate that the symptom complex is characteristic, and has for its basis an infection of the middle ear which extends into the intracranial space along a well defined channel. The ultimate lesion, however, proved to vary considerably in its nature and extent, dependent no doubt on the virulence of the infecting organism and on the extent of its invasion.

Consideration of the many questions which immediately presented themselves in the study of this group led us into an investigation of the numerous and complicated ramifications of the general problems concerned with the acute infectious diseases of early life, and in particular of otitis media and its intracranial complications. Distribution of the cases into two clinical subgroups made it obvious that the causative lesions varied considerably. In one group, constituting the majority, the clinical course was short and the recovery complete, suggesting that the lesion, whatever its nature, was minor and transient. In the second group, the longer duration of the symptoms and their more disabling character were evidences of an anatomic lesion.

A review of the essential clinical features of the cases forming the basis of this study gave rise to certain questions concerning the underlying etiology, pathogenesis and pathology of the symptom complex. Is the acute infection or the otitis media responsible for the cerebral symptoms when both the conditions are present? What is the relationship, if any, between the generalized convulsions observed in cases of acute general infection and the jacksonian seizures in the group of cases under consideration? Is the clinical syndrome presented here characteristic enough to warrant its designation as a clinical entity? Do the early symptoms give a clue to the ultimate outcome in a given case? If the infection in the middle ear is responsible for the cerebral manifestations, what is the pathway of invasion and what is the character of the resultant lesion? Is there a relationship between the severity of the infection in the middle ear and the cerebral symptoms? What is the explanation of the contralateral cerebral involvement observed in some instances? The answer to these questions we shall now consider. A review of the literature aids in clarifying certain features essential for the understanding of the present study.

## REVIEW OF THE LITERATURE

Owing to the lack of direct reference to the problem in hand, a review of the available literature was at first somewhat disappointing. It was found, however, that contributions dealing indirectly with more

general subjects shed light on the subject under consideration. It was necessary to review the literature dealing with convulsions and paralysis in infancy and childhood, that dealing with otogenic encephalitis with its various clinical manifestations and, finally, that concerned with the cerebral symptoms following otogenic thrombosis of the lateral sinus.

*Motor Manifestations Associated with Acute Otitis Media in Infancy and Early Childhood.*—The occurrence of generalized convulsions in acute infections in childhood is so well recognized by physicians as to constitute one of the platitudes of pediatric practice. Somewhat less familiar, but nevertheless recognized for many years, is the occurrence under these circumstances of jacksonian convulsions, often followed by hemiplegia. The problem of hemiplegia in childhood was first given an important place in the literature by Osler's [1] monograph. Most of the cases in Osler's series were apparently of unknown etiology. In some of the cases of infectious origin, otitis media was also present. Gowers [2] expressed the belief that in three fifths of such cases the hemiplegia was of the "primary" type, that is, without recognizable underlying cause, and due to thrombosis of the cerebral veins. In a more recent contribution on the subject, Ford and Schaffer [3] made an exhaustive review of the literature dealing with the etiology of infantile hemiplegia. In 16 of their own series of 75 cases, the hemiplegia was found to be caused by an acute infectious disease, and in 4 of these 16 cases otitis media also was present. In 7 cases the cause was an acute local infection; otitis media was present in 3 of these. In 19 instances, no cause for the hemiplegia could be found, although the febrile state at the onset suggested an infectious etiology. Further reference will be made to this study. In a summary of the general problem of acute hemiplegia in children which is ushered in by a febrile state, Rothman [4] concluded that it does not represent the cerebral form of anterior poliomyelitis, as the original contribution of Strümpell intimated, but that it is merely a syndrome, due either to nonpurulent, localized encephalitis or to vascular lesions.

Other investigators have given more attention to the convulsions occurring in patients with this condition. Lucas and Southard [5] reported

1. Osler, William: The Cerebral Palsies of Children, Philadelphia, P. Blakiston's Son & Co., 1889.

2. Gowers, W. R.: A Manual of Diseases of the Nervous System, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1898, vol. 2, p. 456.

3. Ford, F. R., and Schaffer, A. J.: The Etiology of Infantile Acquired Hemiplegia, Arch. Neurol. & Psychiat. **18**:323 (Sept.) 1927.

4. Rothman, Phillip E.: Polioencephalitis, Am. J. Dis. Child. **42**:124 (July) 1931.

5. Lucas, W. P., and Southard, E. E.: Contributions to the Neurology of the Child: I. Convulsive Tendencies During and after Encephalitis in Children, Boston M. & S. J. **156**:323 (Feb. 29) 1912.

a series of 12 cases of "encephalitis" verified at autopsy during the clinical course of which convulsions were observed. In 3 of the cases otitis media was present. No mention was made of a tendency to lateralization of the symptoms, and no details of the pathologic picture were furnished. Otitis media was not considered a possible cause of the encephalitis or the convulsions. In a series of 490 children suffering from convulsions, Peterman [6] found the seizures in 90 instances to be due to acute infections, including communicable diseases, acute infections of the upper respiratory tract, pneumonia and otitis media. Most of these children (74) were in the first three years of life. This observer made no distinction between generalized and jacksonian convulsions, nor did he mention the occurrence of associated hemiplegia. Levinson,[7] in a report based on a study of 80 patients with acute cerebral manifestations, stated that in 50 the symptoms were transitory. In this subgroup, 9 of the patients had otitis media. In the same subgroup, convulsions were present in 10 instances and hemiplegia in 4, but the author did not associate otitis media with either convulsions or hemiplegia, nor did he discriminate in his report between generalized and jacksonian seizures. In a study dealing more directly with the problem of jacksonian convulsions, Fincher and Dowman [8] analyzed 130 cases, and in 14 found a history of febrile attacks with delirium and convulsions. Of the 14 patients 9 were in the first decade of life. No reference was made to the possible association of jacksonian seizures and otitis media.

A review of the literature dealing with the motor manifestations in early life makes it apparent that in many instances convulsions, particularly those of jacksonian character, may result in hemiparesis or hemiplegia. Many such instances follow an acute infection, at times recognized otitis media. In most of the so-called primary cases, or those without recognized cause, the onset with fever, delirium and other toxic manifestations makes it evident that infection is the basis of the trouble, even though its source is not determined.

*Motor Manifestations in Otogenic Encephalitis.*—In turning to the more closely related field of the intracranial complications of otitis media, it was found that numerous articles in the literature dealt with the subject of otogenic encephalitis and that in some of the cases reported,

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6. Peterman, M. G.: Convulsions in Childhood: Study of Four Hundred and Ninety Cases, J. A. M. A. **99**:546 (Aug. 13) 1932.

7. Levinson, A.: Acute Transitory Cerebral Manifestations in Infants and in Children, J. A. M. A. **101**:765 (Sept. 2) 1933.

8. Fincher, E. F., and Dowman, C. E.: Epileptiform Seizures of Jacksonian Character: Analysis of One Hundred and Thirty Cases, J. A. M. A. **97**:1375 (Nov. 7) 1931.

well defined motor symptoms were described.[9] The question of the possibility of a diffuse or localized inflammatory alteration in the brain consequent to otitis media is of recent origin so far as pathologic substantiation of its presence is concerned, although the concept was suggested by earlier investigators. In some instances, the transitory manifestations of cerebral involvement, such as convulsions or paralysis, following otitis media have been described as examples of "pseudo-abscess." In a report concerned with this subject, Adson [10] described the condition in 3 patients with motor manifestations, in 2 of whom it followed otitis media secondary to acute communicable diseases. In 1 of these 2 patients contralateral facial paralysis of central origin developed, and in the other, motor aphasia and right hemiplegia were the residual manifestations of a generalized convulsion. Both the patients underwent surgical explorations with negative results, and both recovered. Key-Åberg [11] reported the case of a 4 year old child in whom postscarlatinal mastoiditis developed on the right side. After two months, the patient suddenly became comatose; conjugate deviation of the head and eyes to the right appeared, and, the next day, left hemiplegia. An extradural abscess was found. The patient gradually recovered, except for residual left hemiplegia. Cathala and Odinet [12] reported a verified case of acute otogenic encephalitis in a 10 month old child, in whom acute otitis media was followed by unilateral convulsions which later became generalized. Postmortem examination revealed acute diffuse encephalitis. Grinker and Stone [13] reported a series of 13 cases of acute toxic encephalitis, verified by postmortem examination. In 2 of these cases jacksonian convulsions occurred after the onset of acute otitis media. Hlaváček [14] reported the case of a 9 year old child in whom jacksonian seizures occurred in the contralateral

9. In accord with the subject of our thesis, we have limited ourselves to a review of contributions citing the motor symptoms which follow *acute* otitis media. It is recognized that convulsive seizures, more commonly generalized, may follow chronic otitis media, as is illustrated in Romantzeff's cases (Romantzeff, N.: Russk. klin. **8**:67 [July] 1927). In our own experience, the latter complication is not common, is more apt to occur in older children or adults and frequently indicates the presence of a serious cerebral lesion. This condition, however, is obviously not what is concerned in the group of cases under consideration.

10. Adson, A. W.: Pseudobrain Abscess, S. Clin. North America **4**:503, 1924.

11. Key-Åberg, H.: Contribution à l'étude de l'encéphalite otogène, Acta otolaryng. **10**:75, 1927.

12. Cathala, J., and Odinet: Encéphalite aiguë otogène chez le nourrisson, Bull. Soc. de pédiat. de Paris **25**:201, 1927.

13. Grinker, Roy R., and Stone, Theodore T.: Acute Toxic Encephalitis in Childhood: A Clinicopathologic Study of Thirteen Cases, Arch. Neurol. & Psychiat. **20**:244 (Aug.) 1928.

14. Hlaváček, V.: Časop. lék. česk. **2**:1041 (July 11) 1930; cited by Eagleton, W. P.: Intradural Conditions in Relation to Rhinology and Otology, Arch. Otolaryng. **16**:228 (Aug.) 1932.

extremities following an antrotomy for acute otitis and mastoiditis. The author postulated the presence of perivenous infarcts as the cause of the condition. He expressed the belief that if the infarct became infected an abscess or meningitis resulted.

In the otologic literature, therefore, a number of cases are reported in which convulsions, frequently jacksonian, followed by hemiplegia occurred as a consequence of acute otitis media. These and associated cerebral manifestations at times led to the diagnosis of abscess and explorations with negative results were performed in a number of cases. The observers considered them instances of acute otogenic encephalitis. As many of the patients recovered, the exact nature of the underlying process remains uncertain.

*Motor Manifestations Following Otogenic Thrombosis of the Lateral Sinus.*—The contributions already referred to indicate that convulsions, frequently jacksonian, and hemiplegia may occur in the early years of life after a febrile state. At times this symptom complex appears subsequent to an acute communicable disease, with which otitis media has sometimes been associated. Again, otitis media is the only other lesion present. In many of the cases, however, no gross lesion has been discovered to account for the motor symptoms although the etiologic factor had obviously been one of infection. Minor changes in the membrana tympani, now recognized as responsible for serious and extensive complications, have not been mentioned in the reports of these cases.[15]

On the other hand, there are reports of well defined cases of otitis media in which jacksonian seizures and hemiplegia have developed. These symptoms have been attributed to encephalitis resulting from an extension of the infection in the middle ear. The syndrome under consideration, therefore, may occur in the presence of otitis media. In this regard, the articles cited have failed to give a clue to the possible mode of invasion, if indeed, the infection of the middle ear is the source of the trouble. The pathogenesis of the condition is, however, suggested in the part of the literature now reviewed—that concerned

15. That otitis media may exist with but minor local symptoms and signs is a fact that is not new to pediatricians. An important complement to this fact to be constantly borne in mind is that serious, and at times fatal, complications in other portions of the temporal bone and within the intracranial cavity may arise from such "minor" infections of the middle ear. There can be but little doubt that the often scarcely detectable and apparently insignificant changes in the membrana tympani have too often been ignored as a possible etiologic factor in a manifest intracranial lesion. This important matter has been stressed by Love in a recent contribution from this hospital (Love, A. A.: Complications of Otitis Media Without Rupture of the Membrana Tympani, Arch. Otolaryng. **17**:297 [March] 1933).

with the cerebral manifestations of otogenic thrombosis of the lateral sinus.

In 1894, Mischlich [16] reported the case of a patient in whom jacksonian seizures of the contralateral arm appeared in an uncomplicated case of thrombosis of the lateral sinus. In a patient of Hoffmann's,[17] following accidental injury to the wall of the lateral sinus thrombosis developed, which was, in turn, followed by sensory disturbances and weakness of the contralateral arm. Nonne, Oppenheim and Pinneles [18] were cited as having observed symptoms referable to focal lesions of the brain, which included convulsions and paralysis, in association with otogenic thrombosis of the lateral sinus. In some instances, no gross lesions of the brain were present to account for the manifestations. Neff [19] described 2 cases in which motor symptoms developed following thrombosis of the lateral sinus. One patient had weakness of the contralateral side of the face and arm, while the other suffered from jacksonian seizures on the opposite side. Salinger [20] reported thrombosis of the right lateral sinus, revealed at operation in the case of a 9 year old child who had suffered from convulsive seizures followed by left hemiplegia. He cited a similar case which had been brought to his personal attention.

In their monograph concerned with otogenic intracranial complications, Körner and Grünberg [21] reviewed the literature on the cerebral symptoms consequent to thrombosis of the lateral sinus. They asserted that such symptoms may occur without the presence of a gross lesion, and that they are much more common in children and in young adults than in older persons. In Neubauer's case [22] of a 10 year old girl, right hemiplegia and motor and sensory aphasia appeared following thrombosis of the right lateral sinus. These manifestations were ascribed to a toxic influence, or to pressure extending to the left parietal lobe. Mann [23] described a patient in whom weak-

16. Mischlich: Inaug. Dissert., Strassburg, 1894, case 4, cited by Körner and Grünberg.[21]

17. Hoffmann, R.: Ausgedehnte nicht infizierte Thrombose mehrerer Hirnsinus und der Jugularis in Folge einer Operationsverletzung des Sinus transversus, Heilung, Ztschr. f. Ohrenh. **30**:17, 1897.

18. Nonne, Oppenheim, and Pinneles, cited by Bodechtel and Richter.[24]

19. Neff, U.: Beitrag zur Lehre von der otogenen akuten progressiven Osteomyelitis des Schläfebeins beim Kinde und beim Erwaschsenen, Ztschr. f. Ohrenh. **80**:14, 1921.

20. Salinger, S.: Generalized Clonic Spasms, Hemiparesis and Coma: The Result of a Lateral Sinus Thrombosis, Laryngoscope **33**:27 (Jan.) 1923.

21. Körner, O., and Grünberg, K.: Die otitischen Erkrankungen des Hirns, der Hirnhäute und der Blutleiter, ed. 5, Munich, J. F. Bergmann, 1925, p. 97.

22. Neubauer, A.: Nach akuter Mittelohrentzündung aufgetretene Sinusthrombose mit Aphasie und Hemiplegie, Jahrb. f. Kinderh. **54**:361, 1924.

23. Mann: Motorische Aphasie mit gleichzeitiger Lämung des rechten Armes nach linkseitiger otitischer Sinusthrombose, Monatschr. f. Ohrenh. **64**:1207, 1930.

ness of the right arm and motor aphasia developed after ligation of the jugular vein in the treatment of thrombosis of the left lateral sinus. He also cited the case of Ruttin's patient, in whom hemiplegia developed after ligation of the jugular vein. Both these patients recovered. Bodechtel and Richter [24] described a somewhat unusual case in which the motor weakness appeared on the side of the thrombosed lateral sinus. The autopsy revealed an extensive inflammatory softening of the opposite parietal lobe.

It now seems evident that the paracentral manifestations which constitute the symptom complex under consideration not only may follow acute otitis media but, more specifically, may occur in instances of thrombosis of the lateral sinus. If this is the case, the lateral sinus may be one of the channels through which the infectious material passes to reach the parietal lobe, even in those instances in which the sinus is not completely occluded. A clue, therefore, exists to the possible pathway of invasion from the middle ear to the parietal or central region of the cortex. These various suggestions gleaned from the literature will be considered later in the light of our personal observations.

## REPORT OF CASES

As a basis for the present investigation, a series of 23 cases was studied. The patients were seen by us in consultation with members of the staff of the Hospital for Communicable Diseases or with those of the departments of pediatrics and otology. Each patient was examined as to progress on a number of occasions by either one or both of us. In the case of a few of the patients with protracted clinical courses many examinations were made. We have selected from this group 4 characteristic cases, the essential data on the whole group being found in the accompanying table.

CASE 1 (case 13 of table).—*Scarlet fever followed by otitis media in a 2 year old girl. Sudden onset of jacksonian convulsions on the right side followed by right hemiparesis. Subsidence of the symptoms. Appearance two months later of pressure symptoms and slight right hemiparesis. Exploration for an abscess of the temporal lobe, with negative results. Subsequent drainage of an abscess of the parietal lobe with relief of the pressure symptoms. Residual sensory and motor seizures involving the right hand.*

A girl of 2½ years was admitted to the communicable disease wards of the Los Angeles County Hospital on Dec. 4, 1931, with the history of scarlet fever of five days' duration. Four days later, because of bulging ear drums, a bilateral myringotomy was performed with drainage of pus from both the canals. Fifteen days after the myringotomy, a high fever developed, accompanied by vomiting and jacksonian seizures which involved the right extremities and were associated with conjugate deviation of the head and eyes to the right side. Between the

24. Bodechtel, G., and Richter, H.: Ueber Scheitellappensymptome bei otogener Thrombophlebitis der Gegenseite, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:505, 1933.

periods of convulsions, the right arm and leg were flaccid and paretic, and the patient was in a comatose condition. A lumbar puncture revealed bloody spinal fluid. Because a study of the roentgenograms suggested mastoiditis, a bilateral mastoidectomy was performed. The removal of the tegmen antri on each side disclosed a normal dura. A culture of the pus obtained from the mastoids revealed the presence of Streptococcus haemolyticus, which was considered as the probable causative agent.

On the day following the operation, the patient was clinically much improved and was conscious but aphasic. Paresis of the movements of the head and eyes to the right and of the right extremities was present. The clinical symptoms gradually improved, and at the end of five weeks (on Jan. 30, 1932) the mastoidectomy wounds had healed, and the patient was discharged without symptoms.

With the complaint of refusing food and of vomiting on occasion, she was readmitted on February 24 for further study, at which time weakness of the right arm and leg was again noticed. Since evidences of increased intracranial pressure existed, the left temporal lobe was explored, but unsuccessfully, by one of us (C. B. C.). The patient's condition remained unchanged until three weeks later, when, because of increasing drowsiness and irritability, a reexploratory operation was decided on. On March 22, the left temporal lobe was explored farther forward without results. Because of the presence of the weakness of the right side which was most marked in the arm, a trephine opening was made in the left parietal region and the brain explored with a needle. A well encapsulated abscess was encountered at a depth of 3 cm. A small catheter drain was inserted. Pus obtained from the abscess proved to be sterile.

After the drainage of the abscess, the symptoms promptly began to abate. The weakness of the right extremities became less marked, and the aphasia gave way to a transient polyphrasia. The drain was removed within three weeks, and the wound closed without further complications. The patient was discharged as well.

After an interval of five months, however, she was readmitted to the hospital, suffering from spasmodic attacks, preceded by painful sensations, which affected the right arm, and were followed by mental confusion of several minutes' duration. These attacks were observed for a period of eighteen months after the drainage of the abscess, at intervals which varied in duration. When last seen, the patient appeared well except for minor evidences of residual weakness in the right arm. She was, however, very slow in learning to talk.

*Comment.*—This, the first case of the series under consideration, drew our attention to the particular symptom complex of contralateral motor manifestations appearing during a febrile attack and associated with otitis media. It was fortunate that events turned as they did, for an abscess of the parietal lobe might easily have been overlooked.[25]

25. The surgeon was led to explore the parietal lobe as the result of his experience in a previous case, in which the patient died after an unsuccessful attempt had been made to drain an abscess high in the right cerebral hemisphere. At autopsy the brain was studied in the light of the clinical symptoms, which consisted of contralateral hemiparesis, most marked in the arm, and conjugate deviation of the head and eyes to the side of the abscess. Otogenic abscesses of the parietal lobe, however, are not discussed in detail in this connection, and have been reserved for a subsequent study.

*Essential Details of Twenty-Three Cases Presenting Cerebral Manifestations with Acute Otitis Media*

| No. | Age, Years, and Sex | Otitis Media, Side Involved | Duration of Otitis Media Before Onset of Symptoms | Motor Manifestations | Laboratory Findings: White Blood Cell Count | Laboratory Findings: Spinal Fluid and Cell Count | Outcome | Comment |
|---|---|---|---|---|---|---|---|---|
| 1 | 2 F | Right and left | 12 hours | Convulsions on left side with conjugate deviation to left; no residual paralysis | 11,250 | 3 cells | Complete recovery in 16 days | No changes in optic fundus; bradycardia during acute stage |
| 2 | 1 M | Right and left | 4 days | No convulsions or conjugate deviation; right hemiplegia associated with Bell's palsy on right side | 10,900 | 21 cells, mostly polymorphonuclears | Complete recovery in 19 days | Normal fundi; paralysis of movements of shoulders but not of those of elbow or fingers |
| 3 | 1 F | Right and left | 1 day | Generalized convulsions with deviation of head and eyes to the left; residual right hemiplegia | No count | 12 lymphocytes | Complete recovery in 12 days | Veins of fundus overfilled |
| 4 | 11 F | Left | A few hours | Generalized convulsions without conjugate deviation of head or eyes; weakness of lower part of right side of face | 11,200 | Bloody; 80 cells counted later | Complete recovery in 10 days | Veins of fundus overfilled; perivascular exudate |
| 5 | 2 M | Right and left | 3 days | Generalized tremors with convulsive seizures of right arm; no conjugate deviation of head or eyes; right hemiplegia (in arm mostly) | 10,600 | No examination | Complete recovery in 40 days | Normal fundus; marked, prolonged bradycardia; aphasia; later, polyphrasia |
| 6 | 2 F | Right and left | 1 day | Convulsions on right side with deviation of head and eyes to left; residual right hemiplegia | No count | 2 lymphocytes | Complete recovery in 1 day | Normal fundus; bradycardia |
| 7 | 1 F | Left | Immediate onset | Convulsions on right side with deviation of head and eyes to right; residual paralysis of right arm | 12,600 | 4 lymphocytes | Complete recovery in 7 days | Old decerebrate rigidity from injury at birth; normal fundus |
| 8 | 4 F | Right | 3 days | Convulsive seizures of right arm without deviation of head and eyes; no residual weakness | No count | 5 lymphocytes | Complete recovery in 14 days | Choked disks |
| 9 | 2 M | Right and left | 3 days | Seizures on right side, becoming generalized; deviation of head and eyes not ascertained; no residual weakness | No count | No examination | Complete recovery in 10 days | |
| 10 | 2 F | Right | 1 day | Convulsions on left side; no deviation of head and eyes noted; no residual paralysis | 14,000 | No examination | Complete recovery in 10 days | |
| 11 | 6 F | Left | Immediate onset | Generalized convulsions with subsequent deviation of head to left; residual right hemiparesis | No count | 3 lymphocytes | Complete recovery in 14 days | Overfilling of veins of optic fundus |
| 12 | 1 M | Right and left | 1 day | Convulsions on right side affecting the arm most; no deviation; residual weakness in the right arm | 8,200 | Slightly bloody | Complete recovery in 20 days | Slight overfilling of veins in right optic fundus |

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|---|---|---|---|---|---|---|---|---|
| 13 | 2<br>F | Right and left | 15 days | Convulsions on right side with deviation of head and eyes to right; residual weakness of right side | No count | No examination | Recovery; residual hemiparesis and petit attacks | Drainage of an abscess of the left parietal lobe; sterile contents |
| 14 | 4<br>M | Right and left | 7 days | Convulsions on right side, arm most affected; deviation of head and eyes to left during seizures, to the right between them, finally to the right | 40,000 | 150 lymphocytes; pressure of 180 mm. of mercury | Recovery in 5 weeks with residual weakness in hand, difficulties of speech and change in personality | Long period of coma with dissociated eye movements; paralysis of upward gaze, and aphasia |
| 15 | 7<br>F | Right and left | 7 days | Seizures on left side with deviation of head and eyes to left during convulsion; residual weakness in arm and lower part of face on left side | 22,400 | 60 lymphocytes | Recovery in 4 months with residual hemiplegia | Cortical changes in parietal lobe and gliosis of temporal lobe, found at operation |
| 16 | 2<br>F | Right and left* | 2 days | Generalized convulsion with deviation of head to right; residual left hemiplegia | 9,100 | Clear, no cells | Recovery in 4 weeks with residual hemiparesis | |
| 17 | 1<br>F | Right and left | 1 day | Generalized convulsions at first, later on right side, with conjugate deviation to the left, then to the right; residual right hemiplegia | 30,000 | Xanthochromic; 268 cells, mostly lymphocytes | Recovery in 6 weeks with residual hemiplegia | Choked disks |
| 18 | 2<br>F | Bilateral, more on the left | 4 days | Convulsions on right side with deviation of head and eyes to right during seizures, to left later; no weakness apparent | 16,000 | Bloody | Death after 5 days | Autopsy refused |
| 19 | 1<br>F | Left | Immediate onset | Convulsions on right side with conjugate deviation to the right during, and to the left after convulsions; right hemiplegia | 39,000 | 2,010 cells, polymorphonuclears; pneumococci | Death after 1 day | Autopsy revealed pneumococcic meningitis; exudate more abundant over the left cerebral hemisphere |
| 20 | 2<br>M | Right and left | 7 days | No convulsions; deviation of head to left with right hemiplegia | 23,600 | 2 lymphocytes | Death after 2 weeks | Relatively slow pulse; choked disks; at autopsy, no gross lesion of brain seen; microscopic study not made |
| 21 | ½<br>F | Right and left | 7 days | Generalized convulsions with conjugate deviation of head to left during seizure; no residual hemiparesis | No count | At first xanthochromic, later bloody | Death after 11 days | Autopsy revealed generalized streptococcic meningitis; exudate abundant near vault and base |
| 22 | 1<br>M | Right and left (right more pronounced) | 3 days | Convulsions on left side and generalized; no deviation of head or eyes, and no observed hemiparesis | No count | No examination | Death after 4 days | Bacillary dysentery revealed by autopsy; patient was in hospital only a few hours |
| 23 | 4<br>F | Left | 1 day | Convulsions on left side, at times generalized; no deviation of head; tendency to decerebrate rigidity | No count | Clear; 3 lymphocytes | Death after 2 days | Veins of fundus tortuous; death from extensive accidental burns; autopsy revealed marked cerebral edema, more marked on left; hemorrhagic focus in left parietal lobe |

* An acute exacerbation of chronic otitis media.

We were also fortunate in establishing the seat of the lesion so definitely in the parietal lobe.

CASE 2 (case 15 of table).—*Coma and jacksonian seizures on the left side following acute bilateral otitis media. Marked residual left hemiplegia. Bilateral mastoidectomy disclosing marked necrosis of the bony trabeculae on the right. Development of bilateral choked disk. Explorations of the right parietal and temporal lobes for a supposed abscess. Gelatinous appearance of the cortex in the right parietal region and gliosis of the right temporal lobe.*

A 7 year old Mexican girl was admitted to the communicable disease wards of the Los Angeles County Hospital on June 12, 1932, with a history of sudden onset of fever, listlessness, gastro-intestinal disturbances and bilateral earache. Following a fluctuating course of two weeks, the patient became nauseated and vomited. She was brought to the hospital the following day in a comatose state and suffering from convulsions. The seizures involved the left extremities; during an attack, the left arm was adducted and the fist tightly clenched, the left thigh adducted and flexed, the knee slightly flexed, and the toes markedly flexed. The head was deviated to the left. The right extremities remained limp.

Because of the obvious signs of active otitis media, suggesting that the cerebral symptoms were due to an intracranial extension, a bilateral mastoidectomy was performed by Dr. A. A. Love, who found the trabeculae of the right mastoid process to be definitely necrotic while in those of the left only slight changes were evident. For the next few days the patient was extremely irritable, and right hemiplegia developed. The nasal margins of the optic disks were blurred, and there was an elevation of the disk of 1.5 to 2 diopters. These changes subsequently became more marked; the physiologic cups became filled in, and there was an increasing tortuosity of the vessels.

The hemiplegia developed to its most marked extent about two weeks after admission, and then receded slightly during the remainder of the patient's stay in the hospital. The right forearm was sharply flexed on the arm, the thumb and index finger were extended and the other fingers, especially the ring and the little finger, were flexed. A tendency to flexion was apparent at the left knee. Whenever the patient attempted to take a step, the thigh was flexed excessively, so that the foot was raised higher than was intended. It was consequently somewhat difficult to project the foot forward. The rigid extension usually seen in hemiplegia with the characteristic varus posture did not develop. When the patient was placed in a prone position with the paretic upper extremity unsupported over the side of the bed, the forearm automatically extended itself.

With the experience in the previous case and the possibility of an encapsulated abscess in mind, we kept the patient under constant observation with frequent examinations for a period of three months. The possibility of abscess was emphasized by the constant elevation of the white blood count, which ranged between 10,000 and 13,000 cells per cubic millimeter. The diagnosis of an abscess being still unsettled, it was finally deemed safer to perform an exploratory operation than to permit the patient to go home.

With the patient under general anesthesia, a small trephine opening was made in the right parietal region of the skull. The dura was seen to be of normal thickness. The thickened and slightly opaque arachnoid bulged into the small opening in the dura. The underlying tissue could not be clearly distinguished and appeared bluish, owing to the accumulation of fluid which exuded into the wound. After some of the fluid had been evacuated, the cortex appeared some-

what atrophic, yellow and gelatinous. A fine injection of the capillaries in the pia was also present. The lobe was explored with a brain needle and the lateral ventricle encountered at a depth of 4 cm.

Another opening was then made in the right temporal region, just above and behind the auricula at a point situated immediately above the tegmen antri. Here the dura seemed to be thicker than normal, the arachnoid was transparent and the subarachnoid space contained considerable fluid. The cortex of the brain, however, appeared normal, the convolutions were not flattened and no pial congestion was present. The exploring needle met with continued minor resistance in its passage through the lobe, giving one the sensation of its passing through soft cream cheese. The inferior horn of the lateral ventricle was here encountered at a depth of 4 cm.

The patient recovered promptly, and, after the healing of the operative wounds, was discharged on October 5. Hemiplegia was still present to a marked degree.

*Comment.*—With the history of the first case so freshly in mind, we were prejudiced in the belief that in this patient too an abscess might have developed in the parietal region and become latent. Exploration failed to substantiate this suspicion, but did reveal what appeared to be a recent vascular change in the parietal cortex. The change may have been diffuse; at least the persistent and marked hemiparesis led one to suspect this. The change seemed to correspond to the cortical alteration, described by Ford and Schaffner,[3] which was found in the course of an exploratory operation for a similar condition. These observers attributed the alterations to thrombosis, presumably arterial, which was secondary to degenerative changes in the walls of the blood vessels incident to an acute infectious disease. In this case, no evidence of a predisposing acute infection was recognizable. The lesion occurred on the same side as the prominent changes in the mastoid bone. The increased density of the tissue of the right temporal lobe was interpreted as due to gliosis of this lobe consequent to the infection in the middle ear and the mastoid. A similar sensation of resistance is encountered in the tissue of the brain about abscesses of long standing and known to be due to residual gliosis of the region.

CASE 3 (case 12 of table).—*Bilateral otitis media. Repeated jacksonian convulsions on the right side with conjugate deviation of the head and eyes to the left. Transient residual right hemiparesis. Complete recovery.*

An eleven month old white boy was admitted to the communicable disease wards of the Los Angeles County Hospital on Aug. 10, 1933, in a somewhat stuporous condition. The provisional diagnosis was that of acute anterior poliomyelitis. On the afternoon of the day of admission, the patient, who had been well up to that time, was found in a state of tonic convulsion in which the head and eyes were said to have been deviated to the left. Following the convulsive seizure, he remained comatose for forty-five minutes. In the course of the following evening, he had two additional attacks, which began in the right arm but which were associated with deviation of the head and eyes to the left and later to the right. In these subsequent attacks, the patient did not lose consciousness.

On admission, the right extremities were found to be definitely paretic, the arm being predominantly affected. The temperature, which at first was normal, soon rose to 103 F., and the pulse rate increased to 160. The patient was extremely irritable, but lethargic. Because the membrana tympani appeared granular, a bilateral myringotomy was performed by the physician in charge and bloody serum and purulent exudate were obtained. An associated infection of the respiratory tract existed, as indicated by the presence of moist râles in the lungs. The spinal fluid and urine proved to be normal. The white blood cell count was 8,200 per cubic millimeter.

When seen by one of us on the fourth day in the hospital, the patient was found to be very irritable, the eyelids were swollen shut and spastic right hemiplegia was present. The upper extremity was affected more than the lower, and the face was only slightly involved. A slight overfilling of the veins was noted in the right fundus oculi. The abdominal reflexes were absent, and the neck was slightly rigid.

Within a few days, the weakness of the right side began to improve; after twenty days, it had disappeared, and no other symptoms of the illness remained. He was discharged from the hospital on September 9.

*Comment.*—This case was selected as an example of transient hemiplegia following jacksonian convulsions. The conjugate deviation of the head and eyes was somewhat atypical, as compared to that in the other cases, in that the deviation was to the side of the convulsing members at first and away from it after paresis had set in. We interpreted the respiratory infection as responsible for the bilateral otitis media and this in turn as responsible for the extension of the infection into the intracranial space. It seemed most unlikely that in this case an arterial thrombosis, as a result of alterations in the walls of the vessels, accounted for the manifestations, as some observers have suggested.

Although we found that most of the cases in this series presented a fairly uniform clinical picture, it is obvious that all cases cannot be crowded into so circumscribed a category. As is later suggested, considerable variation exists in the distribution and course of the veins draining the cerebral hemispheres. If our conception of the pathway of extension along venous channels proves ultimately to be correct, this variation explains the atypical motor manifestations, and even the more unusual symptoms which at times make their appearance.

The following case is presented to illustrate the occurrence of motor disturbances suggestive of involvement of the brain stem as well as of the cortex.

CASE 4 (case 14 of table).—*Bilateral otitis media following infection of the upper respiratory tract. Jacksonian convulsions on the right side with conjugate deviation of the head and eyes to the left. Residual left hemiplegia of six weeks' duration.*

In a white boy, aged 4½ years, there occurred chills and epistaxis and a convulsion on the right side, followed by weakness of the members of the right side. The forearm remained in extreme flexion with the fingers in the "obstetric" contracture. He had repeated convulsions involving the right side of the face and the

right arm, during which the head and eyes were turned to the left. The patient was in a comatose condition between the seizures.

He was admitted to the hospital on Nov. 20, 1932, in a stuporous state. An examination revealed changes in the membrana tympani, and a bilateral myringotomy was performed, with drainage of pus from both ears. On the day following admission, conjugate deviation of the head and eyes to the right was noted; this was not constant, for at times the eyes wandered about in a blind stare. At other times there was a fixed downward gaze. The pupils measured 1.5 mm. in diameter, but reacted well to light. Signs of a minor meningeal reaction were also present. Use of the right extremities seemed to have been recovered, but the left limbs were immobile.

During the subsequent five days, the patient had two exacerbations of fever; he was forcefully negativistic and injured himself by striking against objects about him. The deep reflexes disappeared, and the pathologic reflexes became evident. Marked dissociation of the eye movements now made its appearance, the two globes moving separately at intervals. A paralysis of upward gaze seemed also to be present. The leukocyte count was 40,000, and examination of the spinal fluid on two occasions revealed, respectively, 150 and 160 lymphocytes. No changes of the fundi were observed during this interval.

This condition continued for three weeks, during all of which time there was a general febrile reaction. This period was followed by one of gradual improvement, with the return of use of the limbs and of full consciousness. No signs of decerebrate rigidity or of the phenomena of Magnus and de Kelijn existed at any time. The patient was discharged on December 29, with a minor residual weakness in the left hand, a tendency to aphasia and changes of personality.

*Comment.*—This patient presented a somewhat unusual condition in the shifting of the convulsive seizures from one side to the other. As the otitis media was bilateral, it is possible that a bilateral extension may have taken place at different times, giving rise to alternating hemiparesis.

It is also possible that venous extension may have taken place into the basilar veins, which at times communicate with those draining the cerebral peduncles. This would account for the dissociation of the eye movements and perhaps as well for the alternating hemiparesis.

## CLINICAL MANIFESTATIONS

The clinical manifestations of the syndrome here described vary within certain limits, but the general type stands out clearly. Its essential characteristics, as illustrated by this group of cases, are as follows:

1. An infant or young child suffers from unilateral or bilateral acute otitis media, often unrecognized, and an acute febrile reaction with leukocytosis develops.

2. Jacksonian convulsions, associated with marked disturbance of consciousness, appear soon after the onset, affecting predominantly the upper extremity. These localized seizures are usually followed by monoplegia brachiofacialis, which may appear, however, in some instances

without the preceding convulsions. Conjugate deviation of the head and eyes is commonly observed during the seizure.

3. In about half of the cases complete recovery occurs in from one to three weeks. In a smaller number recovery is incomplete and residual weakness or contractures result. A few cases terminate fatally. A detailed presentation of the symptoms, as found in the 23 cases which form this series, follows.

*General Data.*—Seventeen of the 23 patients were in the first three years of life. The remaining patients were distributed through childhood, the oldest being 11 years of age. The syndrome occurs particularly in infancy and early childhood. Sixteen of the 23 patients were girls. As there seems to be no logical reason why girls should be predominantly affected, this is probably a matter of chance.

*Otitis Media.*—In 5 cases otitis media occurred on the right side, in 4, on the left side and in 14, bilaterally. This emphasizes the fact that otitis in infancy is frequently bilateral. With the exception of a patient with an acute exacerbation of chronic otitis media, all of the patients were suffering from the acute form. The duration of the otitis before the appearance of convulsions varied somewhat. In 5 cases the convulsions appeared in less than twenty-four hours after clinical evidence of otitis was present, in 7 cases, in about twenty-four hours, and in 4 others, in about three days. In 4 other cases one week elapsed after otitis media had appeared. The history in 3 cases did not conform to these particular periods, but in no case was the otitis present longer than a week.

Another interesting aspect of these cases which merits attention is the rather abrupt onset of the convulsive seizures, which make their appearance shortly after the onset of the febrile state and the otitis media. In most cases they occurred within three days. At times this was almost the first indication that the child was ill. This short interval suggests that vascular invasion took place shortly after the onset of the focal infection, and it may indicate the presence of a more virulent organism. It also precludes the possibility of the development of serious or profound changes in the local mucous membranes, the bone or the dura, so often present in other types of intracranial extension.

In the cases in which the patient recovered it is noteworthy that the average time between the appearance of otitis media and the onset of convulsions was one and one-half days, which was only one-half the time that elapsed in the fatal cases. The question arises whether or not this interval may be taken as a dependable factor in the prognosis of a given case. Only by checking a large number of cases can this be settled.

*Convulsions.*—Convulsive seizures occurred in 22 of the 23 patients. Of this group, only 5 had generalized convulsions, their place in the

group being dependent on other localizing manifestations. Two patients with localized seizures had a history of previous generalized convulsions. This is of interest in that one would naturally anticipate that a potential epileptic patient would react with generalized seizures. In 5 instances both general and local convulsions were observed, the general seizures, as a rule, coming first. Of the 16 instances of jacksonian convulsions, in 11 they were on the right side and in 5, on the left. We have no explanation of the occurrence of the extension twice as frequently from the left ear as from the right. As the otitis media was bilateral in 65 per cent of the cases the laterality of the extension and not the unilaterality of the otitis is the question involved.

*Paralysis.*—Paralysis, characteristically of the brachiofacial type, was present in 17 cases. In 12 of these, it occurred on the right side; in 3, on the left; in 1, it was first on the left, then on the right side, and in another, it was first on the right, and then on the left. In 1 of the last mentioned instances, decerebrate rigidity, due to injury at birth, was present before otitis developed; a typical hemiplegia, nevertheless, developed with the occurrence of the syndrome. The patient died. In 2 cases not included among the 17 just mentioned, the onset of decerebrate rigidity made it impossible to state whether or not hemiparesis actually existed during the height of the symptoms.

*Conjugate Deviation of the Head and Eyes.*—It may be said in general that when conjugate deviation of the head and eyes was present it was coincident with the jacksonian convulsions. In a few cases paralytic deviation toward the opposite side followed. Cases 14 and 17 presented exceptions to this rule, however, since the deviation was toward the side of the lesion during the jacksonian convulsions and away from it afterward. In 1 of these cases, dissociation of the movements of the eye muscles and aphasia developed. The other case had a fatal outcome, but permission for autopsy was refused.

*Changes in the Eyeground.*—An examination of the fundi was made whenever possible—in 20 of the 23 cases. In 9 of these, no changes were present. In 8 others, minor or moderate alterations existed, such as an overfilling of the veins, slight blurring of the margins of the disks or the presence of a slight amount of exudate in the cups. In 3 cases a measurable elevation of the disks was present.

*Changes in the Reflexes.*—In 14 cases the patient was too young for the so-called pathologic reflexes to be significant. In general, the deep and superficial reflexes behaved as one expects in an acute cerebral condition. They were frequently absent during the acute onset and became normal during recovery or increased on the hemiplegic or hemiparetic side. The variable results could not be charted in an intelligible way. Whenever the patients were old enough to have significant pathologic reflexes, these were in harmony with other clinical manifestations.

Meningeal signs were frequent during the acute stage of the illness. Rigidity of the neck, associated with acute otitis media or mastoiditis, as well as Kernig's and Brudzinski's signs, must be discounted, particularly in infancy. In many instances the illness was of short duration and the meningeal signs soon subsided. In some cases no such signs ever appeared.

*Laboratory Findings.*—Blood counts were made in 14 cases. In only 2 was the white blood count below 10,000 per cubic millimeter. In the majority it was over 12,000. In several the count reached nearly 20,000 per cubic millimeter, and in 1 it was 30,000. The examination of the spinal fluid was of interest. In 4 patients definite evidence of blood in the spinal fluid (nontraumatic) was present, and in 1 the fluid was xanthochromic.[26] In 9 instances the spinal fluid was normal in every respect. In 4 of the instances of clear spinal fluid in which the cell count was of value, pleocytosis was noted, the average count being about 30 cells per cubic millimeter. The cells were predominantly lymphocytes. Two cases in which a definite septic meningitis developed were not included in this group. Spinal puncture was not done in 5 cases.

*Duration of the Illness.*—The cases which form the basis for this study may be classified in three groups from the standpoint of the duration of illness: (1) those in which complete recovery occurred; (2) those in which residual symptoms persisted, and (3) those which terminated fatally. In the first group, the duration of the illness varied from one to forty days, the average time being fourteen days. In the second group, the illness lasted much longer, owing in part to the fact that we delayed in pronouncing the patient well, although the acute stage was no longer than in the first group. In the third group, the type of complication determined the duration of the illness. Of the 23 cases here presented, complete recovery occurred in 12 (52 per cent), recovery with residual motor manifestations in 5 (21 per cent) and death in 6 (26 per cent).

*Cause of Death.*—Of the 6 fatal cases, death was due to septic meningitis in 2. One patient died of accidental burns, and 2, of acute cerebral disease. Autopsy was performed in all of the cases except 1 of the 2 in which an acute cerebral lesion was suspected. In this case the brain was incompletely studied by a general pathologist and then discarded. We have been unfortunate, therefore, in not being able to make our own critical study of the brain in an uncomplicated case.

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26. The presence of fresh blood or of xanthochromia in the spinal fluid seems to us further evidence of a venous rather than of an arterial thrombosis. The occurrence of red softening secondary to venous thrombosis often results in the escape of blood into the subarachnoid space. This does not happen in cerebral softening consequent to arterial thrombosis.

PATHOGENESIS

During the later decades of the nineteenth century it was generally assumed that, with the possible exception of septic meningitis, intracranial complications of disease of the middle ear followed the chronic rather than the acute form. For example, Pitt,[27] in reviewing a series of 57 fatal cases of otitis media, stated that the infection must be present for months before an extension into the cranium can occur. With increasing experience in dealing with the various lesions as they were exposed at operation as well as at autopsy, it became evident that, particularly in the early years of life, extension did occur at times during an acute attack of the disease. This is especially true in cases of thrombosis of the lateral sinus, a complication which came to be recognized by surgical means with increasing frequency. Chronic inflammatory changes in the bone and the dura therefore are not necessary for the invasion of the intradural spaces. The only alternative explanation is that of the invasion by way of the vascular channels, which is suggested clinically by an apoplectiform onset of symptoms.

In these cases, did the extension occur by way of the veins or of the arteries? The sudden onset and the localization of lesions at a distance from the affected ear, not infrequently on the opposite side, seemed to favor the conception of spread by way of arterial channels. However, in certain cases of thrombosis of the lateral sinus in which an autopsy was performed, retrograde thrombosis of the afferent cerebral veins or the inferior anastomotic veins was found, with focal lesions at a distance from the original focus in the middle ear. Observations in such instances made it obvious that a distant lesion could be the result of venous extension. However, a short review of the arguments in favor of the theory of arterial spread is in order in this connection.

Infections reaching the brain from the middle ear through the circle of Willis must invade the arterial system either directly, by way of the carotid artery, or indirectly, by way of the general circulation. Direct extension into the carotid artery is unlikely, except in rare instances, for pericarotid infections are very uncommon, and actual otogenic thrombosis of the carotid artery is a pathologic curiosity. Furthermore, even in the recorded cases of thrombosis of the carotid artery, abscesses of the brain or other focal lesions have not been reported, although extensive softening of the homolateral hemisphere has occurred.

The alternate possibility is based on the existence of a general infection with the ultimate lodgment of particles in the brain by way of the arterial tree. In this case again the original atrium of infection is venous, by way of the small radicles draining into the lateral sinus,

27. Pitt, G. Newton: Goulstonian Lectures on Some Cerebral Lesions, Brit. M. J. **1**:643 (March 22) 1890.

resulting in general infection of the blood stream. If this is the method of spread it is reasonable to inquire why no characteristic clinical manifestations of septicemia are present in these instances and why no such peripheral foci of infection occur in the lungs, pleura, joints and skin as are found in the instances of thrombosis of the lateral sinus. Again, if infections of the brain are secondary to a general infection of the blood stream, why is there but one abscess or focal lesion, when as a rule such metastatic lesions are multiple? In order that this concept of the pathogenesis of distant otitic abscesses of the brain be accepted, the case under consideration must meet the established criteria for metastatic abscesses of the brain which are secondary to other types of infection.[28]

When, on the other hand, thrombosis affects all the intervening venous channels between the middle ear and the cerebral focus, the pathway of infection is obvious. The possibility, furthermore, that with a break in this chain of continuous thrombosis, a similar situation may occur has received little consideration. Recently cases have come to light in which thrombosis of the lateral sinus did not occur, although thrombosis of the local veins was present. Still other instances, more frequently encountered, have been found in which thrombosis of the lateral sinus was associated with a focal lesion, particularly in the parietal lobe along the course of the communicating venous channels, which did not appear thrombosed on gross inspection. The literature dealing with this concept has been reviewed earlier in the paper. In following the concept a step further, it must be admitted that when an abscess, for example, is found in the parietal, or even the frontal, lobe, extension by a venous channel occurs either without the intermediation of thrombosis or with thrombosis which had healed in the meantime without residual lesions.[29] Such cases are of the utmost interest in this connection, since they almost invariably occur following acute rather than chronic infections.

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28. In a review of 352 cases of abscess of the brain of otitic origin (Blau, L.: Zur Lehre von den otogenen intrakraniellen Erkrankungen, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ores. **10**:86, 1918), the author found but 6 cases which he thought were due to extension to the brain by way of the arterial tree (metastatic). While all may not agree that some of these cases were not as easily explained on the basis of a venous extension, it may be granted for the moment, for the sake of argument, that they were truly metastatic. With only 6 of a total of 352 cases (1.7 per cent), it seems to be stretching the point to use this explanation for the frequently observed cerebral manifestations secondary to otitis media, such as are found in the group of cases under consideration.

29. A principle which is coming to be more firmly fixed in the minds of those interested in the development of the intracranial complications of otitis media is that certain intermediate lesions, marking the course of invasion, may disappear, whereas the ultimate lesion produced may go on to its full development. In the pathogenesis of cortical changes consequent to otitis media, it is entirely possible that aseptic thrombi in the cortical veins may disappear after obstructing the smaller cortical radicles sufficiently to give rise to gross changes in the brain.

Two difficulties are presented in the acceptance of this explanation—the determination of the actual mode of invasion of the venous system by the noxious agent and the explanation of the presence of lesions in the cerebral hemisphere on the side opposite the draining ear. The actual route by which infection gains entrance to the larger channels is not entirely clear. It is known that the dural veins are larger and present more numerous ramifications in the early years of life. This condition favors the entrance and extension of infection in the venous radicles of the larger dural sinuses and their afferent veins.

A brief review is necessary of the essential anatomy of the communicating channels by which the mechanism of extension is possible. It is to be noted that, while there are certain variations in a number of cases, a general pattern is more or less adhered to.

Some of the numerous small venous channels which drain the middle ear and antrum empty into dural veins which lie above the tegmen tympani and tegmen antri. These, in turn, drain into the lateral sinus in the region of the genu, or into the lateral portion of the superior petrosal sinus. The inferior anastomotic vein (Labbé), the course and vascular connections of which may explain possible widespread or distant inflammatory lesions consequent to otitis media and mastoiditis, is subject to certain anatomic variations. Its course depends largely on the points of its opening into the superficial middle cerebral vein on the one hand and the lateral sinus on the other. At times more than one inferior anastomotic vein may be present. Its superior mouth usually opens into the caudal portion of the superficial middle cerebral vein, although it may open anywhere along the course of the latter. Likewise, the inferior mouth, usually terminating in the region of the genu of the lateral sinus or in the lateral portion of the superior petrosal sinus, may open more posteriorly. The communications of this vessel with the superior anastomotic vein and the afferent radicles of the superficial middle cerebral vein make accessible almost every situation of the dorsolateral surface of the frontal and parietal lobes.

Although the arrangement of the inferior anastomotic veins occurs in most instances as outlined, certain variations should be mentioned. The upper mouth of the inferior anastomotic vein may empty well forward into the middle cerebral vein, making direct connections with the veins which drain the dorsolateral surface of the frontal lobe. The inferior mouth, in rare instances, may open more caudally into the lateral sinus, even approaching the torcula Herophili. In such cases, invasion of this channel by infectious matter from the middle ear is almost impossible. Perhaps of greater significance in the explanation of variable symptoms referable to the brain stem is the occasional drainage of the basilar vein into the superior petrosal sinus, or into the genu of the lateral sinus. When such a situation is present, the cerebral peduncles and the interpeduncular fossa are exposed to the influence of noxious material from the focus in the middle ear.

The actual mechanism of the invasion of this system of veins is not exactly known. The absence of valves in the venous channels, the comparative sluggishness of the blood flow and the easy reversibility of the current, incident to coughing, sneezing, and so on, may well account for the distant localization of an embolus, infected or otherwise,

once it has gained entrance to the lateral sinus or to the lateral portion of the superior petrosal sinus. No doubt, minute particles from the thrombus or clumps of bacteria at times gain entrance to the blood stream and, with a suitable arrangement of the venous channels, may invade this extensive system of channels to reach the dorsolateral surface of the cerebral hemisphere. The occasional widespread involvement of the venous system in infancy and childhood is evidence that this mode of extension is a serious potentiality.

The explanation of contralaterally situated lesions has been, perhaps, the strongest argument in favor of the concept of dissemination of infection by way of the arterial tree. Some of the advocates of the venous avenue of extension have elaborated a rather fanciful explanation for this phenomenon, which has received serious consideration in some circles. The infection is assumed to spread by way of the superior petrosal sinus to the venous plexus of the dorsum sellae, and then to the superior petrosal sinus of the opposite side. The involvement of the dorsolateral surface of the opposite hemisphere necessitates a further extension into the inferior anastomotic vein, a most unlikely development.

Another possibility, strengthened by the pathologic observations in rare instances, is worthy of consideration, although it is obviously not the explanation in the majority of cases. Extension into the inferior anastomotic vein may carry infectious particles to the superior longitudinal sinus. A secondary extension into the superior cerebral veins of the opposite side is supposed to take place, with or without grossly visible thrombosis of these vessels. The chief objection to either of these theories of venous extension is the necessity for the spread to take place either against the normal venous current or across it or both.

It seems to us that a far simpler and more plausible explanation exists particularly in the case of infants and young children. In a previous communication dealing with the fatal complications of otitis media,[30] it was stated that of patients below 2 years of age on whom autopsies had been performed the infection was bilateral in 85 per cent. This is in accord with the observations of Aschoff and Fuld [31] and with general clinical experience. This is often the case, in spite of the very minor changes existing in one of the membranae typani. As further support for the concept that apparently contralateral extension is in fact homolateral extension from a "quiescent" otitis media, we have often found that clinical evidences of intracranial extension have pointed to the ear

30. Courville, C. B., and Nielsen, J. M.: The Fatal Complications of Otitis Media with Particular Reference to the Intracranial Lesions Found in Ten Thousand Autopsies, Arch. Otolaryng. **19**:451 (April) 1934.

31. Aschoff, L., and Fuld, J.: Zur Frage der Otitis media bei Säuglingen und kleinen Kindern., Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **23**:379, 1926.

showing the lesser signs of inflammation, a point which cannot be too strongly emphasized. If these premises are valid, it is likely that lesions apparently arising in the cerebral hemisphere of the side opposite the infected ear are actually secondary to a condition in the contralateral, and apparently uninfected, ear.

If, therefore, the pathogenesis of the clinical syndrome under consideration is determined to be otogenic, the following points seem to us to be fairly well established: (1) Invasion of the brain in infants and young children consequent to acute otitis media not infrequently occurs; (2) the extension into the intracranial space, at least in some instances, is probably by way of the dural veins draining the middle ear, which empty into the genu of the lateral sinus or into the lateral part of the superior petrosal sinus, and thus gain access to the inferior anastomotic vein. This lays open to infection the major portion of the dorsolateral surface of the frontal and the parietal lobe. The point of lodgment is usually within the central region, depending on the arrangement of the veins in each case. When this occurs, the site of predilection, as subsequently shown, is the parietal lobe.

## PATHOLOGIC CONSIDERATIONS

Knowledge of the pathologic character of the entire group of lesions involving the upper cerebrum which make their appearance after the onset of acute otitis media is very meager, owing to the lack of opportunity to study all of the essential features in a given case. Our purpose in this contribution is to present for consideration the lesions which we have observed at operation and at autopsy, in the hope that some steps may be taken leading to the ultimate solution of this important problem.[32]

At the outset one must predicate a number of distinct lesions in view of the wide variability in the duration of the disease. The following classification is suggested, based on the lesions which have come

32. In a discussion of the pathologic basis of the syndrome under discussion, it is essential to make a sharp distinction between the various etiologic factors which are capable of producing similar symptoms in infectious states in children. Attention has already been drawn to the group of cases following acute communicable disease in which the underlying lesion has been proved to be a hemorrhage or an arterial thrombosis. It is unlikely that otitis, by itself, produces such a lesion. We have encountered several instances in which the clinical syndrome and the presence of otitis media suggested that we were dealing with another case of the group under consideration. Postmortem examination revealed in 1 case a cerebral embolism following abortion, and in 2 others, tuberculous meningitis. The cerebral symptoms are due to a localized lesion, which may be any one of many possible types. In one group the lesion is due to acute otitis media. It is important to decide the significance of the various possible etiologic factors in a given case in establishing a clinical diagnosis.

to our attention from our own experience and from a review of the literature:

I. Toxic lesions. These may be local or diffuse, and their mechanism may be on a vascular, inflammatory or irritative basis. In these instances, the short clinical course and complete and rapid recovery indicate that the lesion is transient and curable. The symptoms are probably due to a direct effect on the nerve cells.

II. Organic lesions. These are often present in cases with a prolonged clinical course and persistent residual symptoms.

A. Vascular lesions.

1. Local lesions. These are due to local infarctions and hemorrhages, possibly incident to the occlusion of a venous channel. In spite of a small persistent lesion, recovery may be clinically complete, if the lesion is not located within the motor area.

2. Widespread lesions. These involve more extensive changes in the cortex, resulting in atrophy, distortion and the formation of scars. An associated, extensive gliosis of the underlying white substance may be present. Paralysis may be persistent if the motor cortex is involved.[33]

B. Inflammatory lesions.

1. Meningeal lesions.

(*a*) Focal inflammatory reactions with fibrinous exudation (focal meningitis).

(*b*) Diffuse septic meningitis.[34]

2. Cerebral lesions.

(*a*) Nonsuppurative encephalitis. This is an inflammatory change, probably superimposed on an area of infarction. It may be focal or more widespread.

(*b*) Abscess formation, in which the size and the degree of encapsulation depends on the size of the original focus, as well as on the nature of the invading organism.

*Toxic Otogenic Encephalitis.*—Little is known of the exact nature of this condition. Grinker and Stone [12] described 2 cases of acute toxic encephalitis in childhood with the characteristic syndrome. At autopsy no focal lesions of the brain were noted, but cerebral edema and congestion were manifest. The meninges were thickened and cellular.

33. Such lesions exposed at operation have been reported by Ford and Schaffer,[3] who predicated their presence on the basis of an arterial thrombosis. While this point remains to be proved conclusively, the possibility must be admitted. Hlaváček [13] believed it to be due to perivenous infarction.

34. There has been much discussion as to the possible pathways of septic meningitis secondary to otitis media in infancy and childhood. Most of these cases are primary in the sense that they are due directly to the otitis media, and not to another gross intracranial inflammatory lesion. No good reason exists why meningitis may not be due to an invasion by way of the venous channels as already proposed. The fact that 2 of the patients in our series presented the characteristic symptom complex followed by septic meningitis verified at autopsy makes it appear possible that a causative relationship exists in some instances.

Acute degeneration of the cortical ganglion cells and perivascular oligodendrogliosis were present. Such manifestations are evidently not specific for otogenic encephalitis, since similar changes may be found in any severe general infection. It is not unlikely that they may be more pronounced in the predominantly involved areas to which the clinical symptoms point. This supposition awaits verification. Whether the pathologic changes are due to direct effect of the toxin on the cerebral parenchyma or are secondary to a vascular influence is not known.

*Vascular Lesions.*—According to our conception, the vascular lesions secondary to infection of the middle ear in this group of cases are the result of thrombosis of the affected veins. A recent lesion, therefore, corresponds to the typical hemorrhagic softening consequent to thrombosis of the great sinuses and their afferent vessels. What little is known of the resultant lesion in such cases has been learned from the appearance of the cortex when exposed at operation, sometimes after the acute stage of the lesion. Whether the atrophic, yellowish and gelatinous appearance of the cortex is the result of an arterial or of a venous thrombosis has not yet been established. The characteristics of a lesion resulting from venous thrombosis not being known, doubt still exists as to whether the original lesion causing the scar is due to the obstruction of an artery or of a vein. If this point were established, the problem would be nearer solution.

In 1 of our cases in which the patient was operated on, diffuse cortical changes were observed in the right parietal lobe, associated with local increase in fluid. This picture seems to correspond closely to the changes observed by Ford and Schaffer [3] in their patients at operation. The absence of local inflammatory change indicates that the more diffuse alteration of the cortex was due either to a preexisting vascular lesion or to a diffuse, nonsuppurative inflammatory change which had become quiescent. Observations in such cases doubtless exist which would give a clearer concept of the true character of this type of manifestations.

*Inflammatory Lesions.*—Although a number of verified cases of nonsuppurative encephalitis have been reported, a satisfactory picture of the progress of events in such cases has not been obtained. The relative influence of a vascular lesion, on the one hand, and of a superimposed inflammatory process, on the other, is difficult to estimate, and, indeed, a wide variation may exist in this regard. There is still considerable discussion as to whether true encephalitis ever progresses to form an abscess. Here again, we have little to offer from the standpoint of the definite pathologic changes associated with the symptom complex.

A focal inflammatory lesion of the parietal region was recently noted at autopsy, although unfortunately the clinical history was extremely

meager. From the standpoint of its value in the understanding of the development of inflammatory lesions, the following brief statement of the histologic changes is of interest:

An 11 month old Mexican boy was brought into the hospital in a state near death. Other than the fact that the patient had been sick for three days with a fever, no details were learned of the illness, and a subsequent attempt to secure further information was not successful. Only an incomplete physical examination was made, for he died shortly after admission. At autopsy, bilateral otitis media, with the larger amount of exudate on the right side, was noted. In the left parieto-occipital region, an area of softening was seen from which sections were taken. Microscopic examination revealed this area to be an infarct with complete necrosis of all normally existing elements except the astrocytes. In two separate areas, suppurating foci were observed to extend into the subcortical substance. These foci showed a marginal zone of lymphocytes while the central mass of cells consisted of polymorphonuclears. The surrounding tissue of the brain was hyperemic, and the smaller blood vessels were increased in number. The parenchymatous and interstitial elements could still be distinguished, but were evidently undergoing degenerative changes. In this area could be noted small hemorrhagic foci such as are seen in the so-called red softening of the brain. The small hemorrhages about the vessels suggested the possibility of thrombosis, but no actual thrombi were distinguished. The arachnoid was swollen in some parts, and the pial vessels were markedly engorged.

We interpreted the lesion as one resulting from several foci of suppuration within a rather extensive area of hemorrhagic softening. The loss of morphologic structure in the various elements in this area suggested a process of infarction. Unfortunately, the pathologist failed to make a critical examination of the area for gross vascular lesions.

Two of the patients in the series died of septic meningitis, evidently secondary to the infection in the middle ear. As has already been intimated, the intracranial invasion in such instances may be by way of the venous pathway. This concept was proposed by Hlaváček,[13] who predicated a "perivenous infarct" as the progenitor of either cerebral abscess or meningitis. According to this observer, infarction occurs, and the lesion may then become infected, resulting in an inflammatory lesion, or it may become absorbed and recovery occur.

Abscess formation occurred in but 1 of our cases. A brief review of the clinical history in this case has been given. The abscess, which was successfully drained, appeared after the onset of the characteristic clinical syndrome. A more detailed discussion of the clinical symptomatology and the course of abscesses of the parietal lobe of otogenous origin will be made the subject of further study.

## SUMMARY

1. A characteristic symptom complex is presented in certain cases following an attack of acute otitis media in early life. The sudden onset of jacksonian convulsions, involving particularly the arm and face, often

associated with conjugate deviation of the head and eyes and usually followed by weakness or paralysis of the affected members during a febrile state, suggests a vascular lesion of the cerebrum. The study of a series of 23 cases is the basis for this report.

2. Wide variation in the length of the clinical course is noted in these cases; in some instances, the patient recovers within a week, while in cases with more serious and probably organic changes in the cortex, residual paretic manifestations may persist for weeks or even months after the acute stage of the disease has subsided.

3. The vascular pathway of infection is indicated by the abrupt onset of cerebral manifestations. It is believed that the organisms, their toxins or both gain entrance to the small venous radicles about the middle ear and antrum, then into the local dural veins and ultimately into the dorsolateral venous system by way of the inferior anastomotic vein. The location and extent of the cortical lesion depend on the arrangement of the cortical venous channels, the nature of the vascular lesion and whether or not the lesion becomes infected.

4. The lesion itself may be toxic, thrombotic or infectious. The toxic lesions are transient, and recovery follows within a few days. The lesion is probably an effect of the bacterial toxins on the nerve cells or of the edema of the affected area. Thrombotic lesions, which result in hemorrhagic softening, may be localized or diffuse, depending on their extent. Residual scarring of the cortex probably persists, but if this is slight residual paralysis may not be present. If the lesion is or becomes infected, a diffuse nonpurulent or a localized purulent encephalitis (abscess) results, the character and extent of the lesion depending on the virulence of the infecting organism and the extent of the vascular lesion.

5. A review of the literature reveals reports of a number of cases of the symptom complex under consideration, associated with a febrile state. When no apparent sufficient cause was found, the condition was classified as "primary" hemiplegia. In other cases, the symptoms were attributed to infection of the upper respiratory tract or to a concomitant communicable disease.

6. The symptom complex is a manifestation of the localization of a lesion in the central or parietal area, but it does not indicate the exact nature of the lesion. A similar clinical picture, dependent on hemorrhage or thrombosis following toxic lesions of the arterial walls, may be secondary to acute infectious diseases. In the present series of cases, the vascular lesion is believed to be venous rather than arterial.

# THE FORMATION OF CHARACTER AS OBSERVED IN THE WELL BABY CLINIC

PRELIMINARY REPORT

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Workers in modern well baby clinics have seen the necessity of studying and directing the psychic as well as the physical life of the infant. Habit training, modes of punishment and methods of education are as intrinsic a concern of the well baby clinic as are the regulation of diet and hours of sleep. There is, however, a marked difference in the ability to judge the results and, consequently, to deal with the management in the two fields.

It is comparatively simple to ascertain whether the prescribed regimen is beneficial to the physical development of the infant. One need only observe the weight, height and muscle tone and compare these observations with an average standard. In accordance with the observations the regimen for the particular infant is changed. But what standards are there by which to check the results of treatment in the psychic development of the infant? In fact, is there any one desired average standard in the psychic as there is in the physical life? It is conceivable that one mother would value traits of character in her child which another would scorn. Therefore, before prescribing a method of training the physician should know two general facts: first, what he wishes to achieve and, second, by which method he can best obtain such a result. As one tries to control to a certain degree the physical development of each infant by prescribing cod liver oil for this child and iron for that one, may it not also be possible that one can further or hinder the development of certain traits of character by different methods of training?

At present one is uncertain as to which trait of character one is furthering or retarding when one advises bringing up a child according to this or that method. The modern tendency is to curtail the period of infancy as much as possible: to wean the infant from the bottle

From the Well Baby Clinic, New York Infirmary for Women and Children.

early, to train him in intestinal and bladder control when very young and so on. This modern method, which tends to shorten the period of infancy, is in contrast to the older method, which tends to prolong it. Will the former method lead to the development of different traits of character from the latter? If so, can one and by what means does one observe these differences in the growing infant? If one could ascertain the relationship, if any, between the method of training and the development of certain traits of character, one would be in a position to prescribe a method.

The question naturally arises: To what extent can the pediatrician influence the development of character by prescribing certain types of training in infancy? Are there not other factors in the development of character far more important than habit training, such as the inherited constitution and the parents' emotional stability? These are indeed the two most powerful factors in shaping the psychologic growth of any infant. Unfortunately, the pediatrician has no control over the first and, unless he gives the parents intensive psychotherapy, only a little control over the second. Such intensive treatment requires more time than one can give in the usual well baby clinic, and therefore the pediatrician can contribute in this respect little at best toward the development of character. One must, however, not undervalue the little one can do in this regard, but make use of it to its fullest extent. It is easy to disregard the physician's rôle in the development of character because of its smallness, but the more thought and care given it, the more powerful factor his rôle becomes. The other extreme cannot be accepted, that an infant's character can be completely molded according to demand, and that the form of training is the all-powerful factor. The fact must be accepted that the pediatrician is limited by nature in controlling the inherited constitution, is restricted by time in influencing the parents' emotional problems and is therefore forced to exert his power in the one remaining field, that of training. Because of this fact, habit training must play a rôle greater than that with which it is sometimes credited.

Before studying the effects of either type of training, we considered it advisable to have a control group of infants, one in which habit training was not emphasized, but in which we could make simultaneous observations on the development of traits of character, habits and physical status. Actually, it was impossible not to institute some form of habit training. The modern feeding schedule, every three or four hours, immediately establishes one of the most extensively regulated habits. Even prescribing cereals and vegetables, no matter at what age during the first or second half-year of life, is training. We were forced to conclude that without jeopardizing the physical health of the child: First, it was impossible for us to have an ideal control group

TABLE 1.—*Form of Record of Six Months' Summary of Physical and Psychologic Progress of Infants* *

Name..... Address..... Interviewer.....
No. ..... No. Rooms..... No. Boarders.....
Sex.....Birth Date..... Place..... Type.....Para.....Wt.....
Pregnancy.....
Admittance Age..... Attendance.....

I. Family

| | Birthplace | Age | Years in U. S. A. | Religion | Years Engaged | Years Married | Occupation | Salary |
|---|---|---|---|---|---|---|---|---|
| Father | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... |
| Mother | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... |

| | Physical Health | Relation to Child |
|---|---|---|
| Father | ..... | ..... |
| Mother | ..... | ..... |

Oral:

| | Appetite | Fussy | Vomiting | Alcohol | Chewing | Smoking | Nail Biting | Kissing |
|---|---|---|---|---|---|---|---|---|
| Father | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... |
| Mother | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... |

| | Envy | Optimism | Haste | Restlessness | Attachment | Communicative |
|---|---|---|---|---|---|---|
| Father | ..... | ..... | ..... | ..... | ..... | ..... |
| Mother | ..... | ..... | ..... | ..... | ..... | ..... |

Anal:

| | Constipation | Diarrhea | Enema | Douche |
|---|---|---|---|---|
| Father | ..... | ..... | ..... | ..... |
| Mother | ..... | ..... | ..... | ..... |

| | Stingy | Extravagance | Lacks Initiative | Fastidiousness | Orderliness | Obstinacy |
|---|---|---|---|---|---|---|
| Father | ..... | ..... | ..... | ..... | ..... | ..... |
| Mother | ..... | ..... | ..... | ..... | ..... | ..... |

| | Perseverance | Procrastination | Cruelty | Possessiveness | Conservative | Ambivalent |
|---|---|---|---|---|---|---|
| Father | ..... | ..... | ..... | ..... | ..... | ..... |
| Mother | ..... | ..... | ..... | ..... | ..... | ..... |

Phallic:

| | Enuresis | Urgency | Ambition | Hysterical | Reaction Formation | Acceptance of Own Sex |
|---|---|---|---|---|---|---|
| Father | ..... | ..... | ..... | ..... | ..... | ..... |
| Mother | ..... | ..... | ..... | ..... | ..... | ..... |

Genital:

| | Not Narcissistic | Not Ambivalent | Affectionate | Potency | Frequency of Coitus |
|---|---|---|---|---|---|
| Father | ..... | ..... | ..... | ..... | ..... |
| Mother | ..... | ..... | ..... | ..... | ..... |

Siblings:

| Para | Age | Sex | Grade | Physical | Psychic | Relation to Child |
|---|---|---|---|---|---|---|
| ..... | ..... | ..... | ..... | ..... | ..... | ..... |
| ..... | ..... | ..... | ..... | ..... | ..... | ..... |
| ..... | ..... | ..... | ..... | ..... | ..... | ..... |

II. Other Contacts

| Name | Age | Relation to Child | Remarks |
|---|---|---|---|
| ..... | ..... | ..... | ..... |
| ..... | ..... | ..... | ..... |
| ..... | ..... | ..... | ..... |

III. Physical Date ..... Age.....
Head..... Chest..... Abdomen..... Sitting Height.....
Weight..... Pelidisi..... Tone..... Held head up.....
Length.....
Sat alone..... Stood alone..... Walked alone.....
Recognized: Bottle..... Parent.....
1st tooth..... No. teeth now..... Teething difficulty.....
Outdoors..... Specific word use.....

IV. Illnesses
Rickets..... Anemia..... Others.....
Eczema..... Otitis..... Accidents.....
G.-I. disturbances Operations.....
Vomits..... Convulsions.....
Bowels..... Vacc.....
Special Medications..... T. A. T. .....

* The form has been revised since the article was submitted for publication and is used for six, twelve and eighteen month summaries.

TABLE 1.—*Form of Record of Six Months' Summary of Physical and Psychologic Progress of Infants*—Continued*

V. Feeding

Early feeding difficulties

| | | Age | How | Reaction |
|---|---|---|---|---|
| Weaned from breast † | Began | ..... | ..... | ..... |
| | Ended | ..... | ..... | ..... |
| Stop 2 a. m. ..... | Began | ..... | ..... | ..... |
| | Ended | ..... | ..... | ..... |
| Q. 4 Hrs. ..... | Began | ..... | ..... | ..... |
| | Ended | ..... | ..... | ..... |
| P. C. ..... | Began | ..... | ..... | ..... |
| | Ended | ..... | ..... | ..... |
| Weaned from bottle... | Began | ..... | ..... | ..... |
| | Ended | ..... | ..... | ..... |

Feeding time..... Regular..... Awake for feeding.....
Spoon..... Cup.....
Where fed..... By whom..... With whom.....
Appetite..... Spits..... Hiccup.....
Fussy..... New foods..... Solids.....
Age added cereal..... Veg..... O. J..... C. L. O..... Egg..... Meat.....
Likes cereal..... Veg..... O. J..... C. L. O..... Egg..... Meat.....
Peculiarities.....

VI. Sleep

| | | Hours | Where | Falling Asleep | Requirements | Type |
|---|---|---|---|---|---|---|
| Night | Began | ..... | ..... | ..... | ..... | ..... |
| | Ended | ..... | ..... | ..... | ..... | ..... |
| A. M. | Began | ..... | ..... | ..... | ..... | ..... |
| | Ended | ..... | ..... | ..... | ..... | ..... |
| P. M. | Began | ..... | ..... | ..... | ..... | ..... |
| | Ended | ..... | ..... | ..... | ..... | ..... |

VII. Habit training

Bowel: Natural Regularity..... Time..... No..... Type.....

| | Began and Ended | Type Training | Reaction |
|---|---|---|---|
| Bowel..... | ..... | ..... | ..... |
| Bladder: | | | |
| Day..... | ..... | ..... | ..... |
| Night..... | ..... | ..... | ..... |
| Feeds self.... | ..... | ..... | ..... |
| Excrement interest.... | ..... | ..... | ..... |
| Bath..... | | | |

VIII. Behavior

General disposition.....
Aggression (cries, holding of breath, stubborn)..... Withdrawal.....

| | | Age | What | When | Treatment | Reaction |
|---|---|---|---|---|---|---|
| Fears..... | Began | ..... | ..... | ..... | ..... | ..... |
| | Ended | ..... | ..... | ..... | ..... | ..... |
| Tantrum..... | Began | ..... | ..... | ..... | ..... | ..... |
| | Ended | ..... | ..... | ..... | ..... | ..... |
| Masturbation..... | Began | ..... | ..... | ..... | ..... | ..... |
| | Ended | ..... | ..... | ..... | ..... | ..... |
| Head bang or roll | Began | ..... | ..... | ..... | ..... | ..... |
| | Ended | ..... | ..... | ..... | ..... | ..... |
| Sucks..... | Began | ..... | ..... | ..... | ..... | ..... |
| | Ended | ..... | ..... | ..... | ..... | ..... |
| Punishments and obedience..... | | ..... | ..... | ..... | ..... | ..... |

Play: Toys: Type..... Type of play..... With whom.....

IX. General response to environment

Members of family.....
Adults.....
Children.....
Materials.....

X. Observation on child..... Interviewer.....
Intelligence quotient.....
Left-handed or right-handed.....

XI. Parents' attitude.....

XII. Progress

Child's greatest progress.....
Points at which guidance is needed.....

XIII. Impression (interpretation)..... Interpreter.....

XIV. Treatments.....

† Began training and ended training indicate the number of months required for training.

that had no training, and, second, in the future we would never be able to study the effect of the formation of each habit, but would be limited to the investigation of the habits which we could vary without damage to the welfare of the child.

This is a preliminary report on the so-called control group. The number of infants studied is too few for any definite conclusions. But we feel that the new approach to the study of the formation of character in infants and the method of procedure are sufficiently interesting to warrant publication at this early date (later reports will give our observations on the experimental groups and on this control group followed through adolescence).

## PROCEDURE

The children selected were originally limited to first-born infants and children of similar milieu. However, as this proved impracticable, we eliminated only infants with parents who did not speak English and those who showed obvious mental defects. There were nineteen boys and twenty-six girls, of whom nineteen were first-born, fifteen were second children, five third and one was a fourth child. The families were of different nationalities. The parents of two thirds of the children were both born in the United States, and of the parents of the other third one or both were born abroad. Two thirds of the families were Catholic and one sixth, Jewish, and the rest were of other dominations. The family income varied considerably, the average being $24 a week.

Our program was similar to that followed in many well baby clinics. Breast feeding was encouraged, and complementary or supplementary feedings were added as indicated. The infants had been placed on a three hour feeding schedule while in the obstetric service. This schedule was adjusted to the individual needs. Orange juice and cod liver oil were given as indicated, and solids, such as cereals and vegetables, were added between the third and the fifth month. The infant returned for examination monthly, or more frequently when progress was unsatisfactory. At the interviews we purposely paid little attention to habit training so that we might observe what the mothers did of their own accord with little instruction. When the infant was able to sit, we advised the mother to begin to train the child for the toilet, but gave no details as to method or time. Similar casualness was shown when advising the mother to let the child drink from a cup at about the tenth or twelfth month.

To solve our problems it seemed essential to have a complete cross-section of the child's daily life at various age levels. Our greatest difficulty was to find standard measurements of the psychic life of the infant which could be used as a common factor in comparing different children. In studying the physical development one uses objective measurements, such as weight, height, turgor and the like. In judging the psychic life of the infant who is unable to talk, one must rely on observations of his activities. This method is at best unreliable, the more so when the mother, who finds it particularly difficult to be objective, is the chief source of information.

We divided our findings on the child's daily life into two groups. The first consisted of the notation of facts that could be observed fairly objectively by the mother and could be recorded in figures, such as the number of hours of sleep,

the months of breast feeding and the length of time required to establish habits of cleanliness. The second group consisted of observations of the mother which were influenced by her own attitudes and probably, therefore, were inaccurate. Obviously even the data of the first group were influenced by the mother's reactions, but less than those of the second. Furthermore, the first group of facts, because they could be expressed numerically, lent themselves to comparative study and were helpful in comparing the development of different children.

Summaries could be made only at six month[1] intervals because of the magnitude of the work. They were made out according to the forms shown in table 1. Some of the infants had already passed their first year when the data were collected, so that the mothers' reports may not have been accurate; however, the reports which seemed to be reliable have been included in this preliminary report.

At the monthly visit, the mother was interviewed by a physician from our child guidance staff when possible, otherwise by a psychiatric social worker or a well trained volunteer. We found that we obtained the most accurate data by allowing the mother to give us a detailed account of the infant's daily regimen from rising to going to bed.[2] The mother then felt in no way bound to live up to any expectations or standards which we might set up through questions, and she often told facts which she would otherwise have concealed because of a feeling of guilt. The time required for such an interview was about one hour. We found that the routine ten to twenty minute interviews were too short, not only for collecting the data but for instructing the mother. Many mothers could not grasp so rapidly the verbal and written orders of the physician. Those mothers had been considered to be not cooperative, whereas they really had not understood. It showed us that a well baby clinic in which less than one-half hour is given to each infant does not function at its best. Visits to the home were also made in all cases showing a special problem and in as many others as possible. This procedure seemed to be the most reliable way of collecting data nevertheless it contains many sources of error. Psychologic histories, even when taken by well trained volunteers, are not always satisfactory.

The reactions of the infant while in the clinic were carefully observed and noted; such observations helped to corroborate or correct the mother's statement. The progress of the physical development of the infant was noted from the clinical chart. Unfortunately no mental tests could be given because of lack of funds. We admit that this omission may lead to errors; however, we did not include in the group any infant who showed mental retardation in the usual clinical tests.

## OBSERVATIONS

We hesitate to give statistics, as they are based on so few cases, but since our statistical findings dealing with the physical development of the infant correspond to the generally accepted standards, we think that there may be some value in the other observations. Even though not statistically accurate, the following figures are of interest to those who are carrying on a similar type of work.

1. In the present investigation, monthly or bimonthly records are made, and the six month summaries are compiled from these.

2. Monthly standardized history sheets were not used, for the records were written on blank paper, as is customary in a psychiatric interview.

Group I

1. Feeding

A. Age at which the infant was weaned from the breast

Varied from 1 to 13 months.
In the majority, 3½ months.
In the average case, 4½ months.

B. Age at which the 2 a. m. feeding was omitted

Varied from 1 to 12 months.
In the majority, from 3 to 4 months.
In the average case, 4 months.

C. Age at which the feeding schedule was changed from every three hours to every four hours

Varied from 1 to 10 months.
In the majority, 4 months.
In the average case, 3¾ months.

D. Age at which the infant was weaned from the bottle

Varied from 6 to 14 months.
In the majority, 12 months.
In the average case, 10½ months.

E. Appetite and selectiveness

Five infants had poor, two had fair and one had variable appetite. Eight infants were fussy about food in general and four with regard to specific foods; the five who showed poor appetites were also fussy about their food.

2. Sleep of Infants at the Age of 6 Months

A. Bedtime hour

Ranged from 6 to 8 p. m.
In the majority and in the average case, 7 p. m.

B. Number of hours of sleep at night

Varied from ten to thirteen hours.
In the majority, twelve hours.
In the average case, eleven and one-half hours.

C. Number of hours of sleep in the morning

Varied from one-half to three hours.
In the majority, one hour.

D. Number of hours of sleep in the afternoon

Varied from one-half to three hours.
In the majority, two hours.
In the average case, one and three-fourths hours.

E. Number of hours of sleep in twenty-four hours

In the average case, fourteen and three-fourths hours.

F. Type of sleep at night and where

Five infants slept poorly.

All infants had their own crib in their parents' room.

G. Time of day when the infant slept

Each infant seemed to have its own hour; some started to sleep as early as 7 a. m., and others not until 11 a. m. Similar variation was found in the afternoon time.

3. Habit Training for Cleanliness: Intestinal Control

A. Age at which training began

Varied from 2 to 17 months.

In the majority, 4 months.

In the average case, 6 months.

B. Length of time required to establish the habit

Varied from a few days to eight months.

In the majority, from one to two months.

In the average case, two and one-half months.

Only two infants required more than five months.

C. Relation of the length of time required to establish the habit to age when habit training was begun

Infants whose training was begun during the sixth month required a longer time than those whose training was begun before or after the sixth month.

4. Speech

Owing to the fact that the mothers had to understand what we meant by "speaking one or two words," our data are inadequate. Some mothers considered the mere utterance of "mama" as the beginning of speech; others did so only when "mama" signified one particular person.

5. Physical Development

A. Age at which head was held erect

Varied from 2 to 6 months.

In the majority, 3 months.

In the average case, 3⅛ months.

B. Age at which infant sat alone

Varied from 4½ to 9 months.

In the majority, 6 months.

In the average case, 5½ months.

C. Age at which infant stood alone [3]

Varied from 5½ to 13 months.
In the majority, 10 months.
In the average case, 9 months.

D. Age at which the infant walked alone [3]

Varied from 7 to 14 months.
In the majority, 11 months.
In the average case, 11½ months.

E. Age at which the first tooth appeared

Varied from 4 to 12 months.
In the majority, 6 months.
In the average case, 7 months.

F. Measurements at the age of 6 months

| | Head, Cm. | Chest, Cm. | Abdomen, Cm. | Weight, Gm. | Length, Cm. | Sitting Height, Cm. | Pelidisi |
|---|---|---|---|---|---|---|---|
| Varied from..... | 40 to 47 | 40 to 47 | 38 to 46 | 6,000 to 9,300 | 61.5 to 75 | 41.5 to 51 | 88 to 108 |
| In the majority..... | 42.5 | 43 | 42 | 6,900 | 65 | 43 | 93 |
| In the average case..... | 42.75 | 43 | 42.5 | 7,250 | 66.5 | 44.25 | 95 |

G. Number of hours in the fresh air at the age of 6 months

Varied from an occasional hour to seven and one-half hours.
In the majority, four hours.
In the average case, four and three-fourths hours.

6. Frequency and Types of Disease up to the Age of 6 Months

| Illness | No. of Cases |
|---|---|
| Infections of the respiratory tract | |
| Common colds | 8 |
| Bronchitis | 1 |
| Otitis media | 4 |
| Clinical rickets | |
| Rosary only | 6 |
| Severe | 1 |
| Eczema | 6 |
| Gastro-intestinal disturbances | |
| Diarrhea | 4 |
| Constipation | 5 |
| Vomiting | 4 |
| Thrush | 1 |
| Ringworm | 1 |
| Infection of the prepuce | 1 |
| Anemia | 1 |

3. Mothers took such different standards that it is hard to compare their reports, which accounts for such variations in our observations.

Group II

1. Behavior

These data, the least complete, are not reliable. Mothers are loath to tell of habits, such as masturbation, which they believe to be undesirable or to cast reflection on themselves. Furthermore, the time required to obtain accurate data exceeded the time at our disposal, despite the fact that the interviews lasted about one hour.

A. Thumb sucking in infants under 6 months of age.

Twenty-two babies sucked at one time or another; the thumb was the object of choice; two sucked their toes as well.

Two babies sucked both hands; five the right hand, and six the left hand.

We could not obtain exact data on the other cases.

Methods of correction instituted by the mother:

The most frequent one was removal of the hand from mouth; two put a glove on the hand; two gave pacifiers, and one tied the sleeve.

B. Masturbation

So many mothers denied it that we cannot give figures.

C. Disposition

Eight infants were irritable, cranky or had a bad temper.

Four infants had temper tantrums associated with special activities, as when being dressed, being put to sleep at night or when very hungry.

The deductions from the findings take the following form:

1. Frequency of Physical and Psychic (Behavior and Habit Formation) Deviations from the Average Child

| | No. of Cases |
|---|---|
| Physical deviations | 4 |
| Psychic deviations | 7 |
| Physical and psychic deviations | 7 |
| Total number showing deviations | 18 |

2. Etiologic Factors

A. Economic

B. Physical

Constitutional

Acquired

C. Psychologic

Lack of knowldege in the parents.

Mild emotional difficulties of the parents.

Serious emotional difficulties of the parents.

Family uneducatable because of low mentality or lack of knowledge of English.[4]

4. This entire group was eliminated from the study.

THEORETICAL CONSIDERATIONS

TABLE 2.—*Comparative Record at the Age of 6 Months*

| Family History | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Nationality | | Religion | | Physical Health | | Psychic Health | | Relation to Child | |
| Case No. | Sex | Para Siblings | Grand Parents | Income | F. | M. | F. | M. | F. | M. | F. | M. | F. | M. |

| Feeding | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Age Fed Every 4 Hrs. | Age Stopped 2 A. M. Feeding | Breast Weaned | Awake for Feeding | Length of Feeding | Appetite | Fussy | Vomits | Cup |

| Development | | | | | | |
|---|---|---|---|---|---|---|
| | | Recognizes | | | | |
| Held Head | Sat Alone | Bottle | Parent | Talked | Handedness | Teeth |

| Measurements | | | | | | |
|---|---|---|---|---|---|---|
| Head | Chest | Abdomen | Sitting Height | Length | Weight | Pelidisi |

| Physical | | | | |
|---|---|---|---|---|
| | | | Hours Outdoors | |
| Calories in Diet | Weight Gaining | Illness | A. M. | P. M. |

| Sleep | | | | | | |
|---|---|---|---|---|---|---|
| Hours | | | | | | |
| A. M. | P. M. | Bed Time | Awakens at | Why? | Type of Sleep | Where? |

| Bowel Training | | | | Bladder Training | | | |
|---|---|---|---|---|---|---|---|
| | | | | | | | |
| Began | Ended | Time Needed | Method | Began | Ended | Time Needed | Method |

| Behavior | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Play | | | Habits | | | |
| General Disposition | Aggression | Withdrawal | Fears | Type | Toys | With Whom | What | When | Treatment | Reaction |

| Physicians' Comments | | | | | | |
|---|---|---|---|---|---|---|
| Observation of | | | | | | |
| Mother | Child | IQ | Interpretation | Treatment | Progress | Comment |

We believe that we are approaching the solution of one of our main problems, namely, the method of judging the psychic development of the child. The method of taking the history, the facts investigated, the examination and the method of recording gave us an opportunity to make a comparative study (table 2) of the infant's development. Judgment based solely on the comparative study of the records by one of us corresponded closely to that based on the clinical examination by the pediatrician. This indicated that after the examination of hundreds of infants we should be able to formulate a standard method of examining all infants. Even without experience in phychologic observation a pediatrician can follow this method and is able to formulate a fairly accurate opinion of an infant's development psychically as well as physically.

When we perfect the standard examination we shall be better able to observe and to follow the effect of habit training on the formation of character. In order to appreciate the interrelation, many factors must be considered, three of the most important of which are: (*a*) the method of habit training; (*b*) the age at which the training is begun, and (*c*) the condition of the infant before the training is begun.

(*a*) The method of habit training is of equal or probably greater importance than the age at which the training is begun. The method varies with each mother and depends not only on her attitude toward the specific habit, such as the establishment of regular hours of feeding and sleep, but on her emotional balance, her relationship to her infant and her desire for complete and immediate obedience. The mother with a compulsive neurosis is more insistent that her baby nurse on the minute, regardless of any other factor, such as the infant being asleep or wet or crying. She also is more alarmed if her baby does not finish his entire feeding each time. Consequently she is in a state of tension at feeding hours and creates an atmosphere which acts adversely on her baby. Her compulsive attitude influences unfavorably her relationship to her infant. Naturally the effect of such an unstable mother on her child must be different from that of a well adjusted mother.

Mothers, when left to their own devices, begin training by using such methods as correspond to their own pattern of behavior. For example, we found that several mothers, when trying to establish habits of cleanliness, placed their infants on the combination high chair-toilet chair, where during its meals or play the child could defecate and urinate. In other words, throughout the great part of the day during which the child was seated, it was actually on the toilet. The compulsive neurotic type of mother is more apt to be compulsive throughout all the training of her child. It is no wonder, therefore.

that the babies of such mothers are more prone to exhibit similar neurotic traits. It is too early to give data on this, as we are studying the children as they grow older.

(*b*) The age at which habit training is begun plays an important rôle. In our group, the length of time required to establish control of defecation was longest for those infants whose training was begun in the sixth month. This may well be only a matter of chance. It is, however, interesting to note that different factors are probably involved in training before and after the sixth month. Before the sixth month the important factor is the nurse's ability properly to condition the child, a comparatively simple stimulus-response situation. After the sixth month the child plays a more active part, and the important factor becomes a complex psychologic one, involving the relationship between the nurse or the mother and the child. At the age of 6 months, this object relationship has not become very strong. This may account for the greater difficulty in beginning habit training at this period.

The establishment of bladder control is far more difficult for the infant than is intestinal control. In our group the average age for the development of this habit was between 1 and 2 years, although many infants were only 4 or 5 months old when the habit training was begun. Since the average child does not develop this habit until the second year, one must consider what is the optimum age for starting habit training. First, from the child's point of view, is his nervous system overtaxed when the habit is begun in early infancy? Is a long period of training psychologically detrimental? What is the effect of continued and repeated conflicts between the mother and the child? Second, from the mother's standpoint, is it worth her while to expend so much energy over a long period of time only to save one or two diapers occasionally? Besides, is she not apt to become emotionally upset over these daily struggles?

It is difficult to answer these questions correctly until many more children have been followed from infancy through adolescence.[5] But it hardly can be advisable to force the formation of a habit before the child's nervous system and psychologic adjustment are well developed and stabilized. Krasnogorski has shown that it is possible to establish reflexes only after the age of 3 months. Any training, therefore, before this age is futile, and for several months thereafter lays a questionable strain on the nervous system.

(*c*) The physical and psychologic status of the infant before habit training is begun either facilitates or hinders the development of the

5. This group is being followed through adolescence, and observations are being started on a new group, beginning with the prenatal period.

habit. If the infant has an excellent appetite, sleeps well and is healthy, he can adjust to the four hour feeding schedule more readily than a weakling. Similarly, an infant who has a regular daily hour for his bowel movement can be more easily trained for the use of the toilet. In such cases the establishment of the habit of cleanliness is only a matter of alertness on the part of the mother in catching the stool. It is true that this can hardly be called habit training, in the literal sense of the word, but it is so considered by most mothers. Under such circumstances, the habit can be established in one day. On comparing such an infant with one who has no regular hour for defecation, it is evident that the latter has a far more difficult time and will take longer in developing the habit of cleanliness.

As the physical condition of the infant influences the formation of habits, so too does the psychologic condition, since the relationship between the infant and the mother is the dominating factor in the formation of habits after the intellect has developed. A friendly, loving relationship between the infant and the mother tends to facilitate the development of any habit. The feeling of security which an infant experiences when the same person, mother or nurse, takes care of him is far more favorable for the successful formation of habits than when different people take charge, which often happens with the change of nurses.

The seriousness of any condition cannot be determined from the study of the symptoms alone; one must consider the etiologic factors. Pathologic behavior too frequently is considered the disease entity and treated as such, whereas it is only a symptom of an underlying condition. It is necessary to have the same attitude toward psychologic symptoms as toward physical symptoms. As one physical symptom, like fever, can be the result of such diverse diseases as bronchitis and typhoid fever, so too can psychologic symptoms, such as tantrums, and phobias, be the result of different conditions, for instance, lack of knowledge or emotional instability of the parents.

Before we could, therefore, prescribe for a psychologic symptom, such as not falling asleep when alone, we had to understand the cause of such behavior. We found that when pathologic behavior was a symptom of the parents' emotional instability the prognosis was graver than when it was a result of lack of knowledge. Furthermore, the treatment was very different. If the parents are emotionally unstable, they must be given psychotherapy before we may hope for improvement in the infant's symptoms. If, on the other hand, the infant's pathologic behavior is a result of the parents' lack of knowledge or of a temporary physical disturbance, we expect a relatively good and rapid improvement.

## RESULTS

By this comparative procedure we were able to detect in our group of forty-seven infants six babies, all under 18 months of age, who showed marked behavior difficulties. These might easily have been overlooked in a well baby clinic conducted on the usual noncomparative basis.

Infants rarely showed a deviation in a single activity. When we found deviations in one field of behavior (for example, eating or sleeping) we usually found it in other fields. Frequently one deviation was more pronounced than the others.

Psychologic deviations associated with physical deviations were more frequent than psychologic deviations alone. This confirmed our belief that the psychiatrist for infants and small children should be a pediatrician, or at least work in close connection with the pediatrician. The infant must be treated as a complete physical and psychologic entity.

## CONCLUSIONS

A well baby clinic offers a splendid opportunity to study not only the physical but the psychic development of the infant. This leads to a greater appreciation of the influence that the physical development, constitution, social conditions, parent's attitudes and habit training have on the formation of character, nervous disorders and delinquency. In the majority of infants who showed deviations from the normal, both physical and psychologic factors were involved.

In order to ascertain what influences the modern type of habit training, which tends to shorten the period of infancy, plays in the development of character, it is essential to make a minute study not only of the child's daily life but of his environment and of the emotional stability of the parents.

# CALCIUM AND PHOSPHORUS CONTENT OF THE OFFSPRING AFTER FEEDING VITAMIN D TO THE MOTHER RAT

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AND
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A short preliminary report [1] of this study was made in 1932. In a table presented at that time were shown the amounts of sodium potassium, magnesium, chlorine, nitrogen, calcium and phosphorus in the ash content of rat pups. These figures revealed that the calcium and phosphorus contents of the fetal ash were increased when the mother rat was given vitamin D. In view of the present tendency on the part of obstetricians to give pregnant mothers large doses of vitamin D for various causes, this observation becomes significant, and it seemed advisable to make a further study on a larger series of rat pups, comparing the effect of viosterol and cod liver oil on the calcium and phosphorus transmission from the mother to the fetus.

The rats used for our experimental work were raised from our stock colony. Only first litter pups were used in this study. The female rats of from 110 to 120 days of age were divided into three groups. Ten rats were placed on a basic diet, ten on the basic diet plus viosterol 250 D, and ten on the basic diet plus cod liver oil. The composition of the basic diet, "Bills modification of Steenbock's diet," was as follows: yellow corn 76 parts, crude casein 5 parts, linseed oil meal 16 parts, alfalfa meal 2 parts, an iodized sodium chloride 0.5 part and calcium carbonate 0.5 part. To three parts of the foregoing mixture one part of milk powder was added. On analysis, this diet was found to contain 302 mg. of phosphorus and 418 mg. of calcium per hundred grams.

To the groups receiving vitamin D, the viosterol or the cod liver oil added to the basic diet was administered by pipet in daily doses of 0.12 cc. in the case of viosterol 250 D and of 0.2 cc. in the case of the cod liver oil. This dosage was started as soon as it was determined that the animals were pregnant, and was continued until the end of gestation. It is not to be concluded that this dosage is necessarily the optimum, since we realize that not enough variations were tried in our preliminary

From the Department of Pediatrics, University of Chicago.

1. Swanson, W. W., and Iob, L. V.: Mineral Composition of the Offspring After Feeding Cod Liver Oil and Viosterol to the Mother Rat, Am. J. Dis. Child. **44**:477 (Aug.) 1932.

study. Nicholas and Kuhn,[2] in their experimental work, fed each rat 0.5 cc. of viosterol 250 D. They reported that this dose appeared to inhibit conception. On one-fourth that dose we had a somewhat similar experience in that it was difficult to bring about a subsequent normal pregnancy in the viosterol-fed rats.

## METHODS

Whenever possible, the litters were removed from the cage immediately after birth and before suckling. In the first part of our experimental work, each litter was ashed as a whole. In this further study, more than 300 rat pups were ashed separately in order to obtain sufficient data for statistical treatment. All ashings were done in a muffle

TABLE 1.—*Control Group*

| | | Average per Pup | | | |
|---|---|---|---|---|---|
| Rat | No. Pups per Litter | Moist Weight | Dry Weight | Mm. Calcium per Kilogram Dry Substance | Mm. Phosphorus per Kilogram Dry Substance |
| 299 | 6 | 5.215 | 0.7043 | 577.8 | 781.0 |
| 350 | 11 | 4.989 | 0.7420 | 489.1 | 604.8 |
| 312 | 6 | 6.472 | 1.1059 | 446.0 | 513.3 |
| 318 | 11 | 5.723 | 0.9003 | 415.5 | 560.2 |
| 316 | 8 | 6.220 | 1.0371 | 407.9 | 416.7 |
| 320 | 10 | 5.027 | 0.7983 | 405.5 | 495.6 |
| 303 | 6 | 4.704 | 0.6322 | 494.1 | 585.7 |
| 302 | 9 | 5.914 | 1.0366 | 387.7 | 545.9 |
| 291 | 8 | 5.317 | 0.7827 | 466.2 | 647.5 |
| 317 | 8 | 6.506 | 0.8233 | 625.9 | 724.2 |
| 1 | 12 | 4.686 | 0.6724 | 493.1 | 664.9 |
| 2 | 11 | 5.467 | 0.7952 | 528.8 | 579.9 |
| 5 | 11 | 4.711 | 0.6256 | 505.5 | 706.0 |
| 6 | 9 | 5.280 | 0.7774 | 504.4 | 696.0 |
| 7 | 12 | 4.592 | 0.6079 | 513.5 | 577.0 |
| 8 | 9 | 5.252 | 0.7972 | 510.8 | 663.8 |
| 23 | 12 | 5.079 | 0.6784 | 534.5 | 719.7 |
| 27 | 13 | 4.928 | 0.6501 | 509.9 | 690.2 |
| 32 | 11 | 4.701 | 0.6259 | 506.1 | 660.6 |
| Mean..... | 9.6 | 5.304 | 0.7786 | 490.6 ± 13.8* | 622.8 ± 21.16* |
| Standard deviation..... | | | | 60.1 | 92.2 |

* Errors are standard errors.

furnace at 450 C.; the ash was dissolved in dilute acid and made up to a convenient volume. Aliquots were then taken for duplicate calcium determinations by the method of Fiske and Logan,[3] and phosphorus determinations by the method of Fiske and Subbarow.[4]

## RESULTS AND COMMENT

The values presented in tables 1, 2 and 3 show the figures obtained on the control group of nineteen litters, the viosterol group of fourteen litters and the cod liver oil group of fourteen litters. The data presented in the tables are computed on the average values for a pup in each litter. Space will not permit, nor did the final calculations justify, recording values for each individual rat pup.

2. Nicholas, H. O., and Kuhn, E. M.: J. Clin. Investigation **11**:1313, 1932.
3. Fiske, C. H., and Logan, M.: J. Biol. Chem. **93**:211, 1931.
4. Fiske, C. H., and Subbarow, Y.: J. Biol. Chem. **66**:375, 1925.

TABLE 2.—*Viosterol Group*

| Rat | No. Pups per Litter | Average per Pup: Moist Weight | Dry Weight | Mm. Calcium per Kilogram Dry Substance | Mm. Phosphorus per Kilogram Dry Substance |
|---|---|---|---|---|---|
| 292 | 12 | 5.201 | 0.7000 | 701.9 | 826.3 |
| 300 | 10 | 6.009 | 0.6685 | 534.7 | 654.3 |
| 304 | 9 | 5.522 | 9.8712 | 443.5 | 562.9 |
| 293 | 9 | 5.679 | 0.7570 | 489.1 | 685.5 |
| 21 | 11 | 5.579 | 0.7508 | 584.6 | 748.4 |
| 22 | 13 | 5.181 | 0.7390 | 510.0 | 674.2 |
| 24 | 8 | 4.864 | 0.6904 | 510.7 | 711.1 |
| 25 | 13 | 4.919 | 0.6480 | 529.0 | 727.0 |
| 28 | 12 | 4.773 | 0.7141 | 493.7 | 636.8 |
| 26 | 10 | 4.854 | 0.6522 | 541.1 | 697.2 |
| 29 | 6 | 4.641 | 0.6731 | 503.0 | 700.7 |
| 33 | 8 | 5.785 | 0.8553 | 513.4 | 672.2 |
| 34 | 9 | 4.891 | 0.6586 | 543.9 | 719.6 |
| 38 | 13 | 5.330 | 0.7456 | 582.1 | 728.2 |
| Mean..... | 10.2 | 5.231 | 0.7231 | 534.3 ± 16.3* | 696.0 ± 16.1* |
| Standard deviation..... | | | | 60.8 | 60.3 |

* Errors are standard errors.

TABLE 3.—*Cod Liver Oil Group*

| Rat | No. Pups per Litter | Average per Pup: Moist Weight | Dry Weight | Mm. Calcium per Kilogram Dry Substance | Mm. Phosphorus per Kilogram Dry Substance |
|---|---|---|---|---|---|
| 315 | 10 | 5.676 | 0.9315 | 414.5 | 588.6 |
| 296 | 10 | 5.891 | 0.8154 | 575.2 | 700.2 |
| 311 | 10 | 5.470 | 0.7539 | 598.0 | 753.6 |
| 313 | 10 | 5.424 | 0.8503 | 461.2 | 628.6 |
| 314 | 8 | 5.132 | 0.8572 | 684.2 | 781.2 |
| 9 | 13 | 5.083 | 0.6656 | 516.7 | 713.3 |
| 11 | 9 | 4.950 | 0.7070 | 519.6 | 711.4 |
| 13 | 13 | 4.895 | 0.6554 | 550.3 | 721.0 |
| 14 | 14 | 4.373 | 0.6231 | 550.9 | 738.1 |
| 15 | 11 | 5.043 | 0.7149 | 507.1 | 707.6 |
| 16 | 13 | 5.347 | 0.7099 | 630.5 | 787.0 |
| 20 | 14 | 5.692 | 0.7983 | 567.5 | 766.3 |
| 31 | 14 | 4.792 | 0.7624 | 477.1 | 625.6 |
| 19 | 12 | 5.248 | 0.6990 | 539.1 | 715.8 |
| Mean..... | 11.5 | 5.215 | 0.7531 | 542.3 ± 18.5* | 709.9 ± 15.8* |
| Standard deviation..... | | | | 69.6 | 58.3 |

* Errors are standard errors.

TABLE 4.—*Mathematical Deductions from Experimental Results*

| Groups | Mean: Calcium | Mean: Phosphorus | Difference Between Means: Calcium | Difference Between Means: Phosphorus | Error of Difference: Calcium | Error of Difference: Phosphorus | Difference ÷ Error of Difference: Calcium | Difference ÷ Error of Difference: Phosphorus |
|---|---|---|---|---|---|---|---|---|
| Controls.... | 490.6 | 622.8 | | | | | | |
| Viosterol.... | 534.3 | 696.0 | 43.7 | 73.2 | 21.3 | 26.6 | 2.04 | 2.75 |
| Cod liver oil | 542.3 | 709.9 | 51.7 | 87.1 | 23.2 | 26.4 | 2.23 | 3.30 |

In table 4 we have summarized the mathematical deductions made from the experimental results. In all instances, the difference of the mean divided by the error of the difference is greater than 2. The results, therefore, become significant if the three groups may be considered sufficiently large.

In terms of simple mathematics, it may be stated that the administration of 0.12 cc. of viosterol daily increased the calcium content of the offspring 9 per cent and the phosphorus content 11 per cent. The administration of 0.2 cc. of cod liver oil increased the calcium content 10 per cent and the phosphorus content 12 per cent.

Contrary to the observations of Nicholas and Kuhn,[2] we found that when vitamin D was given to the mothers the phosphorus content expressed in milligrams in the ash of rat pups was on the average greater than the calcium content. In 75 per cent of the 400 duplicate calcium and phosphorus analyses that we have completed on rat pups of the three groups, the ratio of the number of milligrams of calcium to the number of milligrams of phosphorus in the ash is 1 : 1+. Whenever a reversal of this ratio occurs, i. e., when calcium is in excess of the phosphorus, it holds for the entire litter and appears to be a unit trait.

The equivalence of the amounts of viosterol and cod liver oil administered to bring about changes in the ash content is worthy of note. When a comparison is made in Steenbock units, the relation between the dosage of viosterol and that of cod liver oil is 150 : 1.

## SUMMARY

1. The effect of the administration of viosterol or cod liver oil to the mother rat on the calcium and phosphorus transmission to the fetus has been studied on a large number of rat pups.

2. The data obtained show that the ash of rat pups from the group in which the mother rat received 0.12 cc. of viosterol 250 D had a calcium content 9 per cent greater and a phosphorus content 11 per cent greater than the ash of the pups in the control group, and when the mother rat received 0.2 cc. of cod liver oil, the ash of the offspring contained 10 per cent more calcium and 12 per cent more phosphorus than the ash of the offspring from the control group.

# CONTOUR OF THE CHEST IN CHILDREN

## II. ACCORDING TO WEIGHT AND HEIGHT

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Much has been written comparing height, weight and age with various body measurements, but I have found in the literature little comparison of these factors with the thoracic index. Hastings,[1] in 1902, studied a group of children between the ages of 5 and 20 years. He took the average height, average weight and average width and breadth of the chest for each age group. In this way the thoracic index curves prepared from his figures were similar to each other when compared with age, height and weight (table 1).

My report deals with the relationship of the thoracic index to the body weight and height (age was considered in a previous report[2]) of about eighteen thousand Minneapolis school children. The measurements were taken during the winter and spring, 1932-1933.

### METHOD OF MEASUREMENT

In carrying out the measurements the discarding of clothing was limited to stripping to the waist and removing the shoes.

The weight was taken on a portable foot scale, and the readings were taken to the nearest figure in whole numbers.

For the height, a board marked off in intervals of ¼ inch (0.64 cm.) was fastened against the wall. The child placed his heels against the board and stood erect, the head touching the board. To make the reading for height a 12 inch (30.4 cm.) ruler was placed on the head, perpendicular to the measuring board.

### THORACIC INDEX AND WEIGHT

Fifteen thousand, nine hundred and seven children were weighed. The weights were grouped in series of 5 pounds (2.3 Kg.) and the average thoracic index was found for each group (the thoracic index is the ratio of the depth of the chest to the width, with the measurements taken at the level of the nipple). From the graph in chart 1 and table 2 it will be observed that the thoracic index curve in relation to the increase in weight runs parallel to the thoracic index curve in relation to the increase in age. Up to about 50 pounds (22.7 Kg.)

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From the Department of Medicine, University of Minnesota.

1. Hastings, W.: A Manual for Physical Measurements, New York, The Macmillan Company, 1902.

2. Weisman, S. A.: Contour of the Chest in Children: I. According to Age, Am. J. Dis. Child. **48**:502 (Sept.) 1934.

TABLE 1.—*Hastings'*[1] *Figures for Average Thoracic Index, Age, Height and Weight*

| Age, Years | Thoracic Index | | Height, Inches | | Weight, Pounds | |
|---|---|---|---|---|---|---|
| | Boys | Girls | Boys | Girls | Boys | Girls |
| 5 | 706 | 632 | 41.4 | 41.4 | 39.7 | 38.9 |
| 6 | 700 | 687 | 43.6 | 43.3 | 42.7 | 40.8 |
| 7 | 667 | 687 | 45.5 | 45.3 | 47.9 | 45.6 |
| 8 | 656 | 677 | 47.8 | 47.3 | 51.0 | 48.9 |
| 9 | 677 | 677 | 49.6 | 49.7 | 55.3 | 54.9 |
| 10 | 645 | 667 | 51.6 | 51.6 | 61.4 | 59.9 |
| 11 | 645 | 645 | 53.1 | 53.2 | 65.8 | 64.0 |
| 12 | 645 | 667 | 55.2 | 55.9 | 72.7 | 72.9 |
| 13 | 667 | 667 | 56.3 | 58.5 | 78.5 | 83.7 |
| 14 | 667 | 667 | 59.5 | 60.4 | 87.6 | 94.5 |
| 15 | 667 | 667 | 62.3 | 61.73 | 103.4 | 102.9 |
| 16 | 677 | 645 | 65.5 | 62.2 | 116.6 | 111.0 |
| 17 | 733 | 677 | 67.0 | 62.8 | 125.2 | 111.2 |
| 18 | 733 | 677 | 67.4 | 62.9 | 130.6 | 110.6 |
| 19 | 709 | 677 | 67.7 | 63.0 | 136.0 | 113.4 |
| 20 | 709 | 677 | 68.2 | 63.0 | 138.4 | 113.9 |

TABLE 2.—*Relationship of the Contour of the Chest to Weight*

| Weight, Pounds | Number of Subjects | | Average Thoracic Index | | Standard Deviation | | Probable Error | |
|---|---|---|---|---|---|---|---|---|
| | Boys | Girls | Boys | Girls | Boys | Girls | Boys | Girls |
| 25–29 | ... | 1 | ... | 790 | .... | .... | ..... | ..... |
| 30–34 | 8 | 10 | 703 | 727 | 1.83 | 3.01 | ±0.4377 | ±0.6425 |
| 35–39 | 48 | 120 | 709 | 715 | 4.63 | 5.19 | ±0.3122 | ±0.3190 |
| 40–44 | 335 | 417 | 714 | 709 | 4.80 | 4.55 | ±0.1767 | ±0.1497 |
| 45–49 | 549 | 630 | 719 | 712 | 4.34 | 4.37 | ±0.1247 | ±0.1173 |
| 50–54 | 653 | 670 | 696 | 708 | 4.78 | 4.56 | ±0.1161 | ±0.1187 |
| 55–59 | 544 | 649 | 699 | 706 | 3.78 | 4.43 | ±0.1092 | ±0.1173 |
| 60–64 | 750 | 650 | 692 | 708 | 4.31 | 4.40 | ±0.0899 | ±0.1160 |
| 65–69 | 642 | 514 | 692 | 703 | 4.52 | 4.42 | ±0.1110 | ±0.1328 |
| 70–74 | 711 | 476 | 679 | 699 | 4.85 | 4.85 | ±0.1227 | ±0.1497 |
| 75–79 | 477 | 380 | 683 | 701 | 4.71 | 4.89 | ±0.1423 | ±0.1686 |
| 80–84 | 483 | 326 | 697 | 692 | 4.72 | 4.98 | ±0.1425 | ±0.1854 |
| 85–89 | 408 | 335 | 681 | 710 | 4.72 | 4.82 | ±0.1570 | ±0.1773 |
| 90–94 | 334 | 346 | 688 | 692 | 4.57 | 5.34 | ±0.1645 | ±0.1841 |
| 95–99 | 265 | 374 | 691 | 692 | 5.05 | 4.12 | ±0.209 | ±0.1436 |
| 100–104 | 255 | 351 | 689 | 685 | 4.66 | 5.45 | ±0.1962 | ±0.1962 |
| 105–109 | 226 | 355 | 696 | 682 | 5.19 | 5.21 | ±0.1874 | ±0.1827 |
| 110–114 | 225 | 325 | 688 | 684 | 5.28 | 4.98 | ±0.2374 | ±0.1821 |
| 115–119 | 201 | 262 | 688 | 684 | 5.41 | 4.51 | ±0.2589 | ±0.1874 |
| 120–124 | 210 | 188 | 681 | 685 | 4.59 | 5.05 | ±0.2137 | ±0.2480 |
| 125–129 | 154 | 157 | 685 | 685 | 5.38 | 4.99 | ±0.292 | ±0.2684 |
| 130–134 | 159 | 88 | 685 | 685 | 4.67 | 4.84 | ±0.2495 | ±0.3561 |
| 135–139 | 127 | 64 | 681 | 693 | .... | 5.15 | ±0.314 | ±0.4330 |
| 140–144 | 89 | 52 | 683 | 684 | 5.32 | 4.39 | ±0.404 | ±0.4100 |
| 145–149 | 67 | 36 | 675 | 695 | 6.64 | 5.14 | ±0.404 | ±0.5773 |
| 150–154 | 37 | 20 | 668 | 697 | 4.99 | 4.11 | ±0.678 | ±0.6198 |
| 155–159 | 34 | 12 | 690 | 714 | 6.71 | 3.41 | ±0.539 | ±0.6643 |
| 160–164 | 27 | 7 | 678 | 714 | 4.11 | 4.97 | ±0.741 | ±1.254 |
| 165–169 | 18 | 7 | 711 | 691 | 6.66 | 3.87 | ±1.34 | ±0.984 |
| 170–174 | 15 | 7 | 679 | 736 | 11.52 | 3.09 | ±0.607 | ±0.789 |
| 175–179 | 5 | 3 | 646 | 723 | 2.38 | 3.31 | ±1.01 | ±1.288 |
| 180–184 | 6 | 3 | 668 | 729 | 3.03 | 2.88 | ±0.67 | ±1.179 |
| 185–189 | 1 | 4 | 755 | 710 | .... | 5.84 | ..... | ±1.969 |
| 190–194 | 4 | .. | 723 | ... | 2.55 | .... | ±0.67 | ..... |
| 195–199 | .. | .. | ... | ... | .... | .... | ..... | ..... |
| 200–204 | .. | .. | ... | ... | .... | .... | ..... | ..... |
| Total | 8,068 | 7,839 | | | | | | |

in weight the boys have a flatter type of chest. Between 50 and 55 pounds (22.7 and 24.9 Kg.) there does not appear to be much difference between the groups. From 55 to 95 pounds (24.9 to 43.1 Kg.) the boys' chests are definitely flatter than the girls'. At about 95 pounds the girls' chests become more flat than the boys'; this difference con-

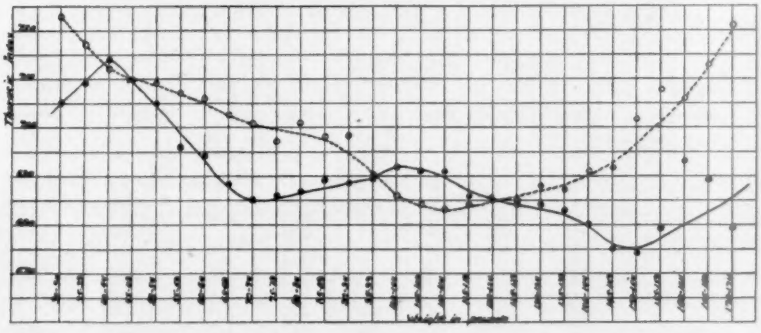

Chart 1.—The thoracic index according to weight, from measurements of 8,068 boys (solid line and circles) and 7,839 girls (broken line and open circles). The curves represent the average thoracic indexes with one application of the three point rule.

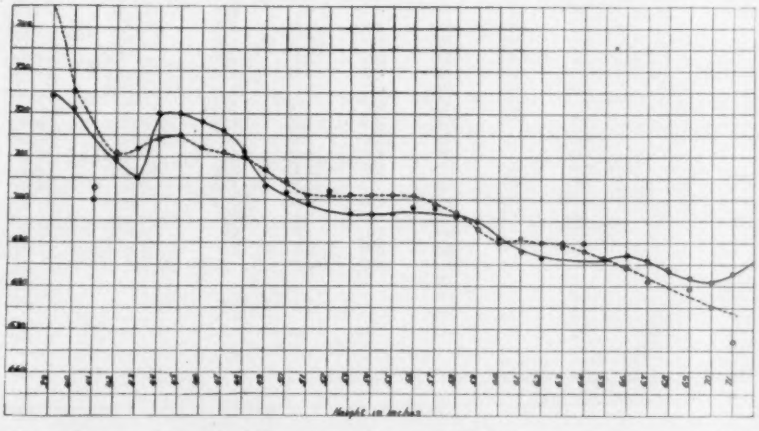

Chart 2.—The thoracic index according to height, from measurements of 8,214 boys (solid line and circles) and 9,407 girls (broken line and open circles). The curves represent the average thoracic indexes with one application of the three point rule.

tinues to about 125 pounds (56.7 Kg.), at which weight the male chest becomes and remains flatter than the female chest. As the weight increases, the contour of the chest tends to flatten. At about 155 pounds (70.3 Kg.) the curve begins to rise rapidly. This, I believe, would

not occur had there been a larger number of cases in this range of weight. Also, a large number of the tall, heavy children were in the older, subnormal group. It should be noted that the thoracic index curve for the boys is more variable than the thoracic index curve for the girls.

TABLE 3.—*Relationship of the Contour of the Chest to Height*

| Height, Inches | Number of Subjects | | Average Thoracic Index | | Standard Deviation | | Probable Error | |
|---|---|---|---|---|---|---|---|---|
| | Boys | Girls | Boys | Girls | Boys | Girls | Boys | Girls |
| 36 | .. | 3 | ... | 745 | .... | 6.14 | ..... | ±2.43 |
| 37 | .. | 3 | ... | 694 | .... | 3.87 | ..... | ±1.537 |
| 38 | .. | .. | ... | ... | .... | .... | ..... | ..... |
| 39 | 5 | 3 | 740 | 776 | 2.7 | 3.87 | ±0.795 | ±1.537 |
| 40 | 11 | 7 | 691 | 685 | 5.14 | 4.91 | ±1.045 | ±1.268 |
| 41 | 23 | 30 | 732 | 714 | 4.01 | 4.81 | ±0.564 | ±0.5899 |
| 42 | 37 | 63 | 676 | 700 | 3.44 | 5.1 | ±0.4565 | ±0.4296 |
| 43 | 87 | 93 | 719 | 709 | 4.68 | 5.91 | ±0.339 | ±0.4148 |
| 44 | 164 | 171 | 721 | 718 | 4.65 | 4.71 | ±0.363 | ±0.2428 |
| 45 | 219 | 214 | 719 | 715 | 4.78 | 4.98 | ±0.217 | ±0.2239 |
| 46 | 264 | 264 | 721 | 712 | 4.57 | 4.64 | ±0.1898 | ±0.1510 |
| 47 | 270 | 311 | 713 | 709 | 4.00 | 4.73 | ±0.1645 | ±0.1800 |
| 48 | 261 | 310 | 713 | 712 | 4.61 | 4.39 | ±0.1902 | ±0.1679 |
| 49 | 348 | 429 | 708 | 710 | 4.78 | 4.88 | ±0.2026 | ±0.1585 |
| 50 | 333 | 376 | 688 | 698 | 3.56 | 4.17 | ±0.1335 | ±0.1450 |
| 51 | 382 | 418 | 711 | 704 | 3.59 | 4.36 | ±0.1221 | ±0.1429 |
| 52 | 384 | 432 | 698 | 700 | 4.14 | 5.50 | ±0.1416 | ±0.1746 |
| 53 | 409 | 396 | 698 | 700 | 4.16 | 4.75 | ±0.1382 | ±0.1598 |
| 54 | 404 | 388 | 695 | 702 | 4.15 | 6.03 | ±0.1389 | ±0.1996 |
| 55 | 409 | 387 | 697 | 700 | 4.57 | 4.15 | ±0.1480 | ±0.1409 |
| 56 | 468 | 361 | 700 | 700 | 4.53 | 4.94 | ±0.1409 | ±0.1733 |
| 57 | 384 | 380 | 696 | 702 | 4.30 | 5.20 | ±0.1477 | ±0.1119 |
| 58 | 431 | 401 | 689 | 696 | 4.28 | 4.69 | ±0.1382 | ±0.1598 |
| 59 | 344 | 401 | 694 | 692 | 4.37 | 4.58 | ±0.1585 | ±0.1537 |
| 60 | 301 | 441 | 693 | 692 | 4.52 | 4.76 | ±0.1746 | ±0.1598 |
| 61 | 252 | 512 | 685 | 687 | 5.09 | 5.07 | ±0.2151 | ±0.1510 |
| 62 | 216 | 547 | 685 | 694 | 5.12 | 4.99 | ±0.2333 | ±0.1436 |
| 63 | 227 | 563 | 690 | 690 | 5.64 | 4.92 | ±0.2676 | ±0.1396 |
| 64 | 190 | 486 | 693 | 687 | 5.65 | 5.02 | ±0.2752 | ±0.1528 |
| 65 | 211 | 370 | 687 | 687 | 5.56 | 4.94 | ±0.257 | ±0.1726 |
| 66 | 202 | 256 | 685 | 683 | 5.10 | 4.78 | ±0.238 | ±0.201 |
| 67 | 250 | 120 | 688 | 683 | 5.51 | 5.57 | ±0.235 | ±0.349 |
| 68 | 194 | 79 | 685 | 677 | 4.90 | 5.35 | ±0.379 | ±0.405 |
| 69 | 225 | 51 | 678 | 688 | 5.45 | 4.80 | ±0.364 | ±0.453 |
| 70 | 122 | 25 | 683 | 671 | 6.22 | 4.44 | ±0.378 | ±0.598 |
| 71 | 85 | 10 | 683 | 665 | 5.80 | 7.14 | ±0.424 | ±1.497 |
| 72 | 70 | 2 | 679 | 648 | 7.42 | 1.0 | ±0.562 | ±0.498 |
| 73 | 17 | 1 | 668 | 729 | 1.9 | 0.0 | ±0.3109 | ±0.9645 |
| 74 | 10 | 2 | 698 | 628 | 5.1 | 2.0 | ±1.08 | ..... |
| 75 | .. | 1 | ... | 680 | .... | .... | ..... | ..... |
| 76 | 2 | .. | 708 | ... | .... | .... | ..... | ..... |
| 77 | 1 | .. | 673 | ... | .... | .... | ..... | ..... |
| 78 | 2 | .. | 709 | ... | .... | .... | ..... | ..... |
| 79 | .. | .. | ... | ... | .... | .... | ..... | ..... |
| 80 | .. | .. | ... | ... | .... | .... | ..... | ..... |
| Total | 8,214 | 9,407 | | | | | | |

## THORACIC INDEX AND HEIGHT

Seventeen thousand, six hundred and twenty-one children were measured. Here, too, with a few exceptions, the thoracic index curve (chart 2; table 3) seems to take a similar course. Up to about 43 inches (109.2 cm.) in height boys have a flatter contour of the chest than girls. Beginning at 44 inches (111.7 cm.) or about the age of 6 years the girls' chests are flatter, and they remain flatter until there is growth to the height of 48 inches (121.9 cm.). From there to

about 58 inches (147.3 cm.) the chest of the average boy is flatter than that of the average girl. Between 58 and 60 inches (152.4 cm.) the girls' chest are again slightly flatter than the boys', but beginning with 61 (154.9 cm.) and up to 65 inches (165.1 cm.) the boys' chests have the flatter contour. At 65 inches the index curves cross again, and from that time the girls' chests are flatter.

In comparing the curves for thoracic index and height with those for thoracic index and weight and age, it is evident that there are some differences, which occur chiefly just before and after adolescence. Generally up to about adolescence boys seem to have the flatter contour of the chest; and, with the exception of the period of height from 59 to 60 inches, the contour remains flatter until the height is about 65 inches. At this point the difference is most marked. The girls' chests seem to stay flatter; while in the correlations of the thoracic index with weight and age the boys have a flatter type of chest than the girls. The same general characteristics, however, are outstanding in the study of the thoracic index in relation to height as were found in the study correlating the thoracic index with age and weight, namely, that as height increases the thoracic index tends to decrease.

## SUMMARY AND CONCLUSIONS

A study correlating weight and height with the thoracic index is presented.

As weight increases there is a tendency toward flattening of the chest.

The increase in the thoracic index at a weight of about 150 pounds and more, as shown in the study, may be due to the much smaller number of cases studied in these groups.

As height increases the thoracic index tends to decrease.

Variations in the thoracic index at different stages of development are also presented.

# CONTOUR OF THE CHEST IN CHILDREN

## III. ENVIRONMENT

S. A. WEISMAN. M.D.
MINNEAPOLIS

Reports that there is a difference in physical development between children reared in poor and those reared in favorable social environments have appeared in the literature for over a hundred years. In 1829, Villermé [1] of France showed that proper food and good homes aid materially in physical development. Roberts,[2] in 1878, after measuring several thousands of the male population in England, showed that boys from the higher social strata were heavier and taller. One year later, Bowditch,[3] of Boston, concluded that persons in the so-called "favored classes" were superior in height and in weight to those in the "general classes." Later, in 1913, Young [4] reported that in Chicago the children of wealthy parents were taller and heavier and had greater vital capacities than the average public school child. Likewise, Schlesinger,[5] Wurzinger [6] and Martin,[7] in Germany, showed that there was a definite difference in stature between the children of the upper classes and the children of the working people.

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From the Department of Medicine, University of Minnesota.

1. Villermé, L. R.: Mémoire sur la taille de l'homme en France, Ann. d'hyg. **1**:351, 1829.

2. Roberts, C.: Manual of Anthropometry, London, J. & A. Churchill, 1878, p. 118.

3. Bowditch, H. P.: The Growth of Children, Rep. Bd. Health Massachusetts **10**:33, 1879.

4. Young, J. E.: Supernormal Environment in Its Relation to the Normal Child, Tr. Internat. Cong. School Hyg. **2**:17, 1913.

5. Schlesinger, E.: Unterschiede im Wachstum bei Schulkindern und jungen Leuten von verschiedener Konstitution und aus verschiedenen Bevölkerungsschichten, Deutsche med. Wchnschr. **43**:1607, 1917.

6. Wurzinger, Stephan: Habitustypen und Körperentwicklung im Schulalter nach Studien an 510 Münchener Volksschülern, Ztschr. f. d. ges. Anat. (Abt. 2) **13**:715, 1928.

7. Martin, R.: Die Körperentwicklung Münchener Volksschulkinder in den Jahren 1921, 1922, 1923, Anthrop. Anz. **1**:76, 1924.

The purpose of the observations to be reported here was to study the contour of the chest of children from the "best" sections of Minneapolis and to compare the measurements with those of the contour of the chest of children from the poorest sections.

In order to carry out the comparison four representative schools in the best section and four in the poorest section were selected. In each section over two thousand children were measured.

In the first section were included the John Burroughs and the Eugene Field schools, which are in the best neighborhoods, the Sidney Pratt school, which is in the University of Minnesota district, and the Calhoun school, which is in a good district, near a large, outlying business district. In the other section were the Washington, Jackson and Sumner schools, which are in the poorest districts, both economically and socially; most of the people in the districts are said to be supported by charity. The Harrison school was also included. This is in a neighborhood somewhat better than the other three schools, yet rather poor.

## THORACIC INDEX

The thoracic index is the ratio between the depth and the width of the chest. In comparing the thoracic indexes a separate curve was drawn for each school (charts 1 and 2 and tables 1 and 2). It should be observed that there is a marked difference between the curves of the thoracic index of the children in the schools in the best communities and the curves of the children in the schools in the poorest neighborhoods, the former curves being the lowest, thus showing flatter, or better developed, chests. It is interesting to note that the children in the schools in the poorest districts of Minneapolis, the Washington and Sumner schools, had definitely the highest curves, showing those children to have the highest thoracic indexes or the deepest, most rounded chests. The best curves, showing the lowest thoracic indexes or the flattest chests, were those of the two schools in perhaps the best district in the whole series, namely, the John Burroughs and the Eugene Field.

There being such a definite difference in the contour of the chest between the children in the poorer and those in the better districts, an investigation was carried out regarding the possible differences in height and weight. These measurements, too, appear to show appreciable differences between the groups.

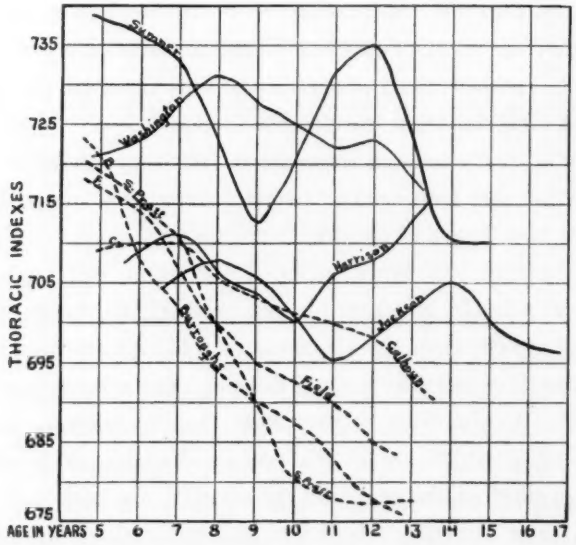


Chart 1.—The thoracic index, according to age, for boys, is shown in curves of the medians for whole school groups. The curves for 781 boys from a poor environment are represented by the solid lines, and those for 1,171 boys from a good environment, by the broken lines. The curves were obtained by one application of the three point rule.

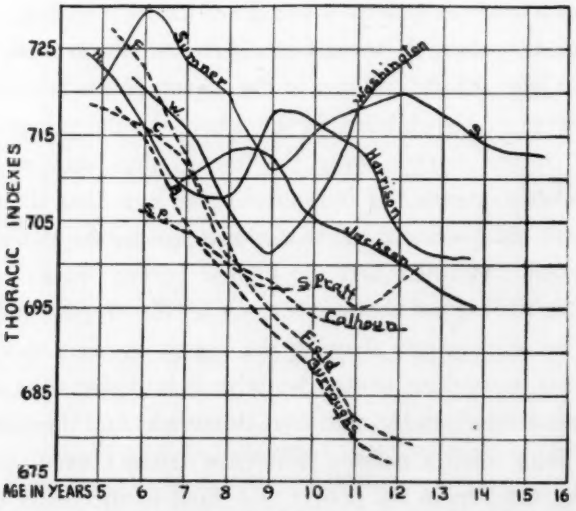


Chart 2.—The thoracic index, according to age, for girls, is shown in curves of the medians for whole school groups. The curves for 878 girls from a poor environment are represented by the solid lines, and those for 1,139 girls from a good environment, by the broken lines. The curves were obtained by one application of the three point rule.

TABLE 1.—*Median Thoracic Indexes for Boys* *

| School | Age, Years | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 |
| Washington† | | | | | | | | | | | |
| Subjects | 15 | 9 | 25 | 29 | 22 | 11 | 23 | 23 | 14 | 5 | 1 |
| Thoracic index | 714 | 736 | 719 | 729 | 745 | 707 | 718 | 737 | 709 | 684 | 683 |
| Harrison† | | | | | | | | | | | |
| Subjects | 9 | 22 | 57 | 48 | 54 | 45 | 46 | 18 | 7 | 1 | |
| Thoracic index | 736 | 707 | 714 | 711 | 694 | 707 | 700 | 712 | 681 | 773 | |
| Sumner† | | | | | | | | | | | |
| Subjects | ... | 15 | 24 | 21 | 15 | 8 | 14 | 9 | 17 | 20 | 8 |
| Thoracic index | ... | 743 | 732 | 722 | 714 | 700 | 745 | 750 | 711 | 691 | 712 |
| Jackson† | | | | | | | | | | | |
| Subjects | 8 | 8 | 15 | 15 | 19 | 19 | 15 | 23 | 32 | 32 | 20 |
| Thoracic index | 743 | 671 | 706 | 711 | 700 | 692 | 690 | 712 | 704 | 699 | 649 |
| Field‡ | | | | | | | | | | | |
| Subjects | ... | 49 | 49 | 29 | 32 | 53 | 37 | 23 | 1 | 2 | 1 |
| Thoracic index | ... | 714 | 714 | 694 | 692 | 700 | 690 | 681 | 674 | 716 | 658 |
| Calhoun‡ | | | | | | | | | | | |
| Subjects | 35 | 44 | 54 | 56 | 73 | 77 | 67 | 42 | 14 | 5 | |
| Thoracic index | 706 | 704 | 710 | 706 | 703 | 700 | 700 | 700 | 695 | 721 | |
| Burroughs‡ | | | | | | | | | | | |
| Subjects | 23 | 33 | 33 | 21 | 49 | 39 | 24 | 10 | | | |
| Thoracic index | 722 | 714 | 694 | 692 | 690 | 692 | 679 | 679 | | | |
| Pratt‡ | | | | | | | | | | | |
| Subjects | 8 | 16 | 22 | 32 | 19 | 28 | 13 | 21 | 23 | 11 | 3 |
| Thoracic index | 716 | 725 | 705 | 704 | 690 | 675 | 675 | 683 | 675 | 667 | 756 |

* In the better districts 1,171 boys were measured, and in the poorer districts 781 boys were measured.
† A school in one of the poorer districts.
‡ A school in one of the better districts.

TABLE 2.—*Median Thoracic Indexes for Girls* *

| School | Age, Years | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 |
| Washington† | | | | | | | | | | | |
| Subjects | 13 | 10 | 19 | 28 | 24 | 30 | 18 | 17 | 6 | 3 | |
| Thoracic index | 719 | 723 | 736 | 706 | 718 | 709 | 720 | 725 | 707 | 743 | |
| Harrison† | | | | | | | | | | | |
| Subjects | 15 | 34 | 55 | 62 | 47 | 45 | 43 | 27 | 6 | 2 | |
| Thoracic index | 727 | 714 | 706 | 706 | 711 | 737 | 700 | 704 | 706 | 683 | |
| Sumner† | | | | | | | | | | | |
| Subjects | ... | 14 | 20 | 25 | 24 | 12 | 18 | 14 | 11 | 17 | 23 |
| Thoracic index | ... | 716 | 727 | 706 | 689 | 707 | 725 | 721 | 714 | 714 | 714 |
| Jackson† | | | | | | | | | | | |
| Subjects | 8 | 8 | 21 | 20 | 9 | 17 | 10 | 21 | 34 | 24 | 24 |
| Thoracic index | 681 | 773 | 706 | 718 | 714 | 703 | 683 | 711 | 714 | 694 | 681 |
| Field‡ | | | | | | | | | | | |
| Subjects | 1 | 51 | 36 | 24 | 49 | 45 | 44 | 21 | 3 | | |
| Thoracic index | 736 | 727 | 714 | 697 | 700 | 684 | 691 | 675 | 674 | | |
| Calhoun‡ | | | | | | | | | | | |
| Subjects | 31 | 31 | 39 | 61 | 52 | 76 | 71 | 38 | 4 | 3 | 1 |
| Thoracic index | 714 | 727 | 706 | 703 | 697 | 694 | 692 | 693 | 711 | 744 | 684 |
| Burroughs‡ | | | | | | | | | | | |
| Subjects | 16 | 36 | 35 | 30 | 41 | 61 | 23 | 13 | | | |
| Thoracic index | 710 | 727 | 694 | 693 | 711 | 675 | 684 | 667 | | | |
| Pratt‡ | | | | | | | | | | | |
| Subjects | 4 | 13 | 29 | 28 | 28 | 30 | 19 | 20 | 20 | 7 | |
| Thoracic index | 671 | 709 | 700 | 701 | 694 | 694 | 703 | 687 | 705 | 675 | |

* In the better districts 1,139 girls were measured, and in the poorer districts 878 girls were measured.
† A school in one of the poorer districts.
‡ A school in one of the better districts.

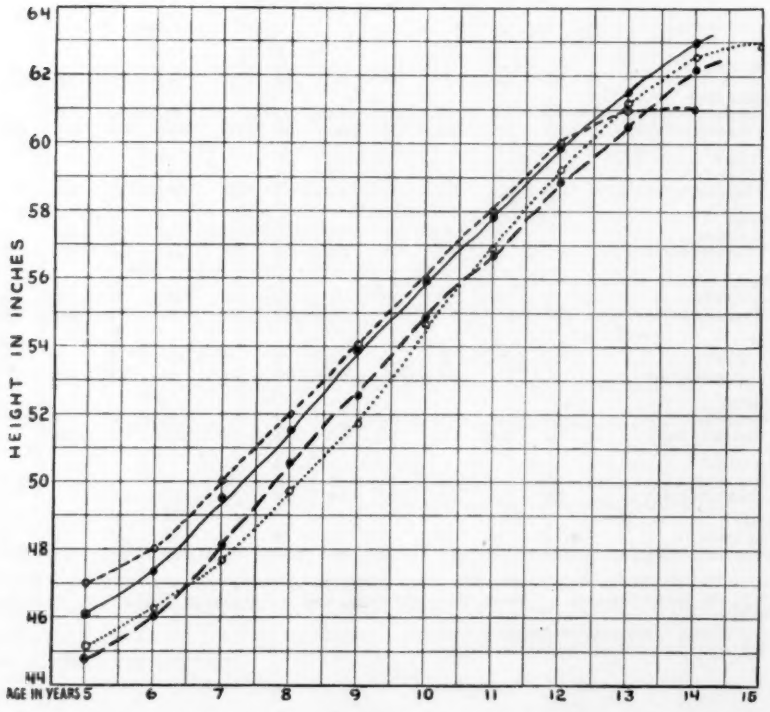


Chart 3.—Curves showing median height, according to age, illustrating the environmental differences in development. The curves for 1,058 boys from a poor environment is represented by the long dashes, and that for 1,226 girls, by the dotted line. The curve for 1,159 boys from a good environment is represented by the unbroken line, and that for 1,146 girls, by the short dashes. The curves were obtained by one application of the three point rule.

TABLE 3.—*Measurements of Height*

| Age, Years | Children From Better Districts* | | | | Children From Poorer Districts† | | | |
|---|---|---|---|---|---|---|---|---|
| | Boys | | Girls | | Boys | | Girls | |
| | Subjects | Median | Subjects | Median | Subjects | Median | Subjects | Median |
| 5 | 62 | 45.6 | 51 | 47.0 | 50 | 44.3 | 50 | 44.8 |
| 6 | 140 | 47.7 | 133 | 47.0 | 79 | 45.8 | 89 | 46.4 |
| 7 | 160 | 49.0 | 137 | 50.0 | 60 | 48.3 | 139 | 48.1 |
| 8 | 137 | 52.0 | 143 | 52.0 | 153 | 50.9 | 170 | 48.6 |
| 9 | 170 | 53.8 | 170 | 54.0 | 156 | 52.5 | 155 | 52.4 |
| 10 | 197 | 56.0 | 223 | 57.0 | 114 | 54.3 | 132 | 54.4 |
| 11 | 138 | 58.0 | 158 | 58.0 | 141 | 57.7 | 128 | 57.2 |
| 12 | 97 | 59.5 | 93 | 59.9 | 107 | 58.2 | 126 | 59.2 |
| 13 | 37 | 62.0 | 27 | 61.0 | 110 | 60.9 | 107 | 61.8 |
| 14 | 21 | 63.0 | 11 | 61.0 | 88 | 62.8 | 84 | 62.7 |
| 15 | .. | .... | .. | .... | .. | .... | 46 | 63.0 |

* The total number of measurements of boys from the better districts was 1,159, and of girls, 1,146.

† The total number of measurements of boys from the poorer districts was 1,058, and of girls, 1,226.

HEIGHT

Chart 3 and table 3 show that the children from the best districts were, on the average, taller than the children from the poorest districts. In both groups the girls were taller than the boys up to the time of puberty, at which time the height of the boys in both groups tended to overtake that of the girls.

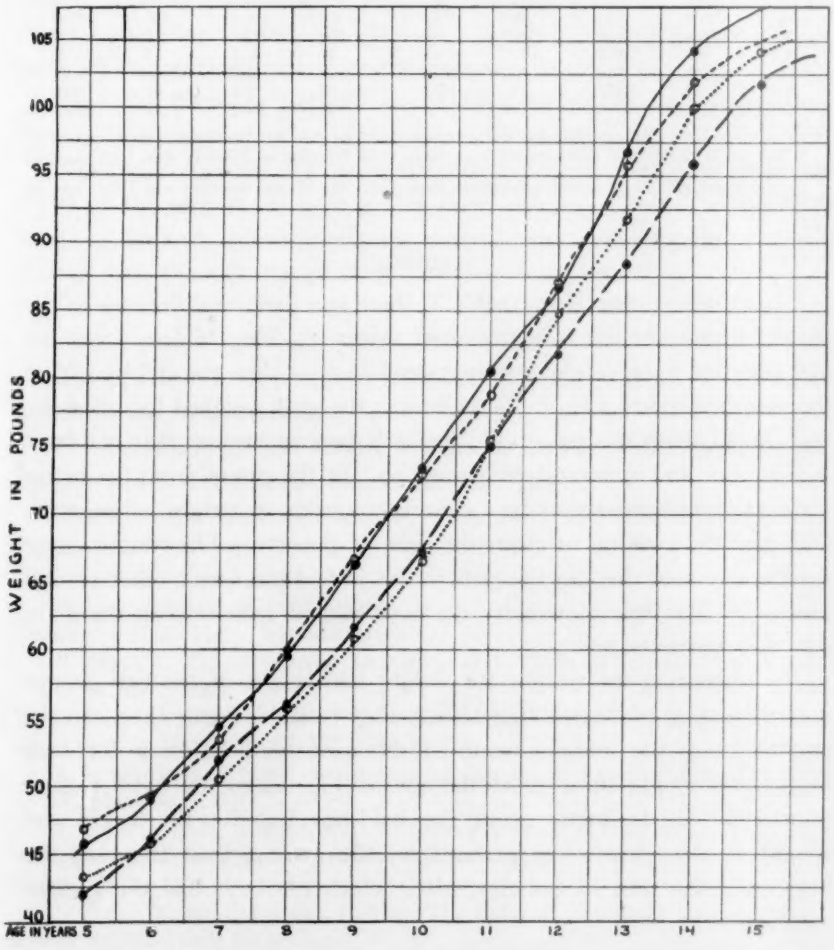


Chart 4.—Curves showing median weight, according to age, illustrating the environmental differences in development. The curve for 1,073 boys from a poor environment is represented by the long dashes, and that for 1,003 girls, by the dotted line. The curve for 1.105 boys from good environments is represented by the unbroken line, and that for 1,087 girls, by the short dashes. The curves were obtained by one application of the three point rule.

TABLE 4.—*Measurements of Weight*

| Age, Years | Children From Better Districts* | | | | Children From Poorer Districts† | | | |
|---|---|---|---|---|---|---|---|---|
| | Boys | | Girls | | Boys | | Girls | |
| | Subjects | Median Lb. | Subjects | Median Lb. | Subjects | Median Lb. | Subjects | Median Lb. |
| 5 | 62 | 44.5 | 50 | 46.0 | 50 | 40.0 | 53 | 42.1 |
| 6 | 128 | 48.7 | 128 | 48.0 | 66 | 46.4 | 74 | 44.8 |
| 7 | 159 | 53.0 | 132 | 53.0 | 134 | 51.9 | 118 | 50.8 |
| 8 | 125 | 60.5 | 137 | 59.4 | 133 | 58.0 | 174 | 55.8 |
| 9 | 164 | 65.0 | 166 | 66.7 | 137 | 58.7 | 130 | 60.2 |
| 10 | 190 | 75.4 | 158 | 75.5 | 106 | 69.0 | 120 | 67.5 |
| 11 | 135 | 81.0 | 193 | 76.9 | 122 | 75.2 | 73 | 74.6 |
| 12 | 89 | 86.0 | 87 | 83.0 | 99 | 81.4 | 75 | 84.8 |
| 13 | 36 | 96.9 | 27 | 103.5 | 95 | 89.2 | 95 | 95.2 |
| 14 | 17 | 108.0 | 9 | 102.5 | 69 | 93.2 | 67 | 98.8 |
| 15 | .. | .... | .. | .... | 62 | 106.3 | 24 | 106.6 |

* The total number of measurements of boys from the better districts was 1,105, and of girls, 1,087.

† The total number of measurements of boys from the poorer districts was 1,073, and of girls, 1,003.

## WEIGHT

As shown in chart 4 and table 4, there is a definite difference in the weight curves for the two groups of children. The children from the best districts were, on the average, much heavier than the children from the poorer districts. In the latter group, the girls weighed less than the boys up to about the age of 11; the girls became heavier than the boys at that time and apparently remained so. In the group from the better districts there appeared to be a close relationship in weight between the boys and the girls up to about the time of puberty. The weight curve for the boys and that for the girls in this period cross each other several times. At the time of puberty the boys became heavier than the girls, and they remained so.

In comparing the graphs for weight and height of the two groups it is interesting to observe how closely they resemble each other. In the poorer group the measurements of the girls were inferior for both height and weight until about the age of 11. Then the boys' picture improved. In the better group the tendency was for the height and weight of the girls to be perhaps a little greater than those of the boys until the time of puberty, after which the boys had the greater measurements.

## POSSIBLE ETIOLOGIC FACTORS

The marked differences in the contour of the chest, the height and the weight of the children of the two groups may indicate that, in addition to poor housing and undesirable hygienic conditions, improper food greatly influences the factors that bring about these differences.

It has been shown that vitamin A promotes growth and builds up resistance to infection.[8] The children of the poorer classes generally do not consume enough milk, butter, eggs and green vegetables, all of which are rich in vitamin A. On the other hand, they eat the less expensive foods, such as lard and vegetable fats and oils, which contain no vitamin A.

Bloch,[9] Ross,[10] McCarrison,[11] Wilson and DuBois,[12] Hume and Smith,[13] Tilden and Miller,[14] Fletcher and Graham [15] and many others have shown that the lack of vitamin A is a contributing factor to diseases of the respiratory and gastro-intestinal tracts.

May not then the lack of proper food be one of the outstanding factors in the maintenance of the infantile, primitive chest?

Finally, proper dietary instructions to expectant mothers may prove to be of the utmost value in giving their children a better start. The work of Dann [16] on lower animals suggests the possibility that vitamin A may pass through the placenta into the fetal circulation.

## CONCLUSIONS AND SUMMARY

The physical development of school children taken from two extreme social groups is compared.

On the average, the children from the better social group were heavier and taller than those from the poor group.

The children from the better environment had the flatter type of chest.

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8. Eusterman, G. B., and Wilbur, D. L.: Clinical Features of Vitamin A Deficiency, J. A. M. A. **98**:2054 (June 11) 1932.

9. Bloch, C. E.: Effects of Deficiency in Vitamins in Infancy, Am. J. Dis. Child. **42**:263 (Aug.) 1931.

10. Ross, S. G.: Nutritional Keratomalacia in Infants, with Report of Four Cases, Am. J. Dis. Child. **22**:232 (Sept.) 1921.

11. McCarrison, Robert: Some Surgical Aspects of Faulty Nutrition, Lancet **1**:1151 (May 23) 1931.

12. Wilson, J. R., and DuBois, R. O.: Report of a Fatal Case of Keratomalacia in an Infant, with Postmortem Examination, Am. J. Dis. Child. **26**:431 (Nov.) 1923.

13. Hume, E. M., and Smith, H. H.: The Value of Foodstuffs for Vitamin A, Lancet **2**:1362 (Dec. 20) 1930.

14. Tilden, E. B., and Miller, E. G., Jr., quoted by Sherman and Smith: The Vitamins, ed. 2, New York, Chemical Catalog Company, 1931.

15. Fletcher, A. A., and Graham, D.: The Large Bowel in Chronic Arthritis, Tr. A. Am. Physicians **44**:231, 1929.

16. Dann, W. J.: The Transmission of Vitamin A from Parents to Young in Mammals, Biochem. J. **26**:1072, 1932.

# SYMPTOMATIC THERAPY IN MALIGNANT DIPHTHERIA

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In the past ten years malignant diphtheria has increased alarmingly in Europe and has occasionally assumed epidemic proportions, with a mortality rate of from 30 to 60 per cent, in spite of the administration of large doses of antitoxin. Attempts to explain the high death rate by identifying a new strain of the diphtheria bacillus have been unsuccessful. Furthermore, accompanying infections with other organisms do not seem to play a deciding rôle in the development of malignant diphtheria.[1] The suggestion that the high rate of mortality was due, in great part, to the physiologic disturbances produced by the diphtheria toxin before the neutralizing effect of antitoxin was obtained, and that therapeutic success required the adequate symptomatic treatment of these disturbances was responsible for the studies reported in the earlier papers of this series.[2] In the present article the treatment of malignant diphtheria from the physiologic point of view is discussed. While the number of severe cases of diphtheria seen in the New Haven Hospital has been small, our clinical experience illustrates certain practical procedures the application of which may benefit patients with the disease.

In previously published reports [2] experimental data were presented which indicated that diphtheritic intoxication produced extensive derangement of carbohydrate metabolism, manifested by changes in the blood sugar and amino-acid nitrogen and by diminished hepatic

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1. Hottinger, A.: Ueber die maligne, sogenannte toxische Diphtherie, Berlin, S. Karger, 1932.

2. Yannet, H., and Darrow, D. C.: Physiological Disturbances During Diphtheritic Intoxication: I. Blood Sugar, Lactic Acid and Non-Protein and Amino-Acid, J. Clin. Investigation **12**:767 (Sept.) 1933; II. Hepatic Glycogenesis and Glycogen Concentration of Cardiac and Skeletal Muscle, ibid. **12**:779 (Sept.) 1933. Yannet, H., and Goldfarb, W.: Physiological Disturbances During Diphtheritic Intoxication: III. Respiratory Quotient and Metabolic Rates, ibid. **12**:787 (Sept.) 1933. Darrow, D. C.; Yannet, H., and Cary, M. K.: Physiological Disturbances During Experimental Diphtheritic Intoxication: Blood Electrolyte and Hemoglobin Concentration, ibid. **13**:553 (July) 1934.

glycogenesis. Moreover, increased hemoglobin concentration, diminished serum electrolyte concentration and azotemia were demonstrated. These observations correlated well with the anatomic changes in the tissues and were interpreted as indicative of hepatic insufficiency, renal insufficiency and shock. In the articles reporting these experimental observations it was pointed out that analogous changes have been noted in patients with malignant diphtheria.

## THERAPEUTIC CONSIDERATION

It is unnecessary to recall the clinical picture of malignant diphtheria, since it is well described in textbooks and in a recent review by Hottinger.[1] However, it may be helpful to call attention to the disturbances that seem likely to dominate the picture during the various phases of the disease. The collapse occurring during the first week of malignant diphtheria is undoubtedly related to the generalized injury to the tissues produced by the diphtheria toxin, in which hepatic insufficiency, capillary damage, myocardial failure and nephritis all play rôles. The application of the term "early cardiac failure" to the stage of collapse is unfortunate. By focusing attention on the heart necessary consideration has been diverted from other important aspects of the disease. Although myocardial failure may occur during the first week, it is more frequently met with after the seventh day. After the second week complications arising from paralyses may dominate the picture.

In the treatment of malignant diphtheria three difficulties demand special attention: (1) vomiting, (2) myocardial failure and (3) maintenance of nutrition.

*Vomiting.*—Persistent vomiting is usually followed by death. The cause of vomiting is not clear, but it is practically always accompanied by enlargement of the liver, which at times may be due chiefly to hepatic necrosis and after the tenth day is usually due to passive congestion. The vomiting must be stopped or minimized not only because of the strain on the heart but in order to avoid loss of electrolytes and resulting dehydration. For this purpose cessation of all feeding by mouth as long as nausea or vomiting persists is practically imperative. Later we shall illustrate that it was possible to supply sufficient fluid intravenously and subcutaneously to sustain for eleven days a patient who had constant nausea and retching during that period.

Physiologic solution of sodium chloride and solutions of dextrose, administered by hypodermoclysis and intravenously, temporarily fulfil the electrolyte and water requirements and supply an appreciable quantity of easily assimilable carbohydrate to tide the patient over this period of enforced starvation. For hypodermoclyses saline or modified alkaline

saline solutions are employed. When it seems advisable to give less salt in relation to water than is contained in physiologic solution of sodium chloride a mixture of equal parts of physiologic solution of sodium chloride and a 5 per cent solution of dextrose may be injected subcutaneously. Because a 5 per cent solution of dextrose alone attracts electrolytes and water from the body before being absorbed [3] it can produce toxic symptoms if injected subcutaneously in large amounts. The combined saline and dextrose solution is more satisfactory. In calculating the amount of fluid to give parenterally an allowance must be made for the imperceptible perspiration as well as for the fluid necessary for the formation of urine.

In general, when dealing with young children, in whom the veins available for intravenous therapy are limited, we have confined ourselves to the use of a 10 per cent solution of dextrose. Concentrated solutions (over 25 per cent) tend to cause thrombosis, and when infiltration of the subcutaneous tissue accidentally occurs, they may even, in rare cases, produce sloughs. It is important to point out that when the fluid is given slowly over a period of from two to three hours comparatively large quantities can be administered intravenously without danger in the presence of frank cardiac decompensation. In from two to three hours we have given as much as 250 cc. to children aged 2 years, and as much as 600 cc. to children aged from 5 to 6 years. Although we have, as yet, had no occasion to utilize infusion by the constant drip method, it is a valuable means for the safe administration of large quantities of fluid. In the case of older children, with easily accessible veins, we have injected from 30 to 50 cc. of a 50 per cent solution of dextrose daily, or more often, during this stage of the disease.

In discussing the parenteral administration of fluid, we refer particularly to the severe cases, in which vomiting is invariably present. In milder cases, fluids rich in carbohydrate or fortified by the addition of dextrose should be given by mouth.[4]

*Myocardial Failure.*—The use of digitalis in the cardiac failure associated with diphtheria has been the source of much controversy.[5] In selected cases, in appropriate dosages and combined with the aforementioned symptomatic therapy, it may prove of great value, as shown by

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3. Schechter, A. J.; Cary, M. K.; Carpentieri, A. L., and Darrow, D. C.: Changes in Composition of Fluids Injected into the Peritoneal Cavity, Am. J Dis. Child. **46**:1015 (Nov.) 1933.

4. Benn, E. C.; Hughes, E., and Alstead, S.: Toxic Diphtheria: Combined Antitoxin and Dextrose-Insulin Therapy, Lancet **1**:281, 1932. Kostyol, L.: Ueber ein neueres Heilverfahren der Diphtherie, Monatschr. f. Kinderh. **48**:162, 1930.

5. McCulloch, H.: On the Administration of Digitalis to Children with Diphtheria, South. M. J. **14**:110, 1921.

our experience and that of others.[6] Bush[7] and Gold[8] have shown experimentally that animals which have received injections of diphtheria toxin become more susceptible to the action of digitalis. It is possible that some of the previous unfortunate experiences have been due to failure to appreciate this fact and to the absence of adequate electrocardiographic study throughout the period of the administration of digitalis. Although von Bokay[9] uses digitalis almost as a prophylactic by administering the drug in small doses daily, beginning with the third or fourth day, we believe that digitalis should be administered only in the presence, and not in anticipation, of congestive cardiac failure. Because of the variability in individual susceptibility it is almost impossible to state the exact amount that should be given. Moreover, because of the combination of intoxication with congestive cardiac failure it becomes exceedingly difficult to use the clinical picture alone as an indication of the adequate effect of digitalis. For these reasons, frequent electrocardiographic study is almost imperative, and daily or more frequent records may be required. An initial dose of 10 mg. of dried digitalis leaf per kilogram, followed in twelve hours by one-half this amount, the latter dose being repeated every twelve hours, has been found to be a useful guide.

The necessity for complete freedom from exertion and the use of small doses of morphine to allay restlessness are too well known to require emphasis.

*Maintenance of Nutrition.*—As indicated, the presence of nausea precludes the administration of food or water by mouth. The effects of starvation are minimized somewhat by the subcutaneous and intravenous injection of dextrose. Transfusions may also be of service in this respect.[10] The edema associated with a low content of serum protein which sometimes occurs following prolonged starvation in cases of diphtheria is of relatively little importance so far as the immediate

6. (*a*) Bie, V., and Schwensen, C.: Effect of Digitalis on Two Cases of Arrhythmia in Diphtheria, J. Infect. Dis. **30**:308 (March) 1922. (*b*) Cathala, J., and Boegner, M.: Traitement du syndrome secondaire de la diphtérie maligne chez l'enfant, par les injections intra-veineuses d'ouabaine, Bull. Soc. de pédiat. de Paris **27**:138, 1929. (*c*) Lesne, M., and Waitz, M., in discussion on Cathala and Boegner.[6b]

7. Bush, A. D.: Drug Action as Modified by Disease Toxins; Ouabain Versus Diphtheria Toxin, J. Pharmacol. & Exper. Therap. **13**:55, 1919.

8. Gold, H.: Tolerance to Digitalis in Experimental Diphtheria, J. A. M. A. **87**:2047 (Dec. 18) 1926.

9. von Bokay, J.: Die Diphtherie seit Bretonneau, Ergebn. d. inn Med. u. Kinderh. **42**:463, 1932.

10. Benedict, H.: Die Behandlung maligner Diphtherie mit Bluttransfusionen, Klin. Wchnschr. **11**:1549, 1932. Dimmel, H.: Bluttransfusionen bei maligner Diphtherie, Med. Klin. **29**:1578, 1933.

welfare of the patient is concerned. It rapidly disappears as soon as feeding is resumed.

Palatal paralysis may offer difficulties in feeding at a time when food can be assimilated if it can be safely administered. Because of the prolonged period of starvation to which some patients may have to be subjected, it may be expedient to resort to gavage, although the dangers incident to this procedure in the presence of probable epiglottal paralysis should be appreciated. An unfortunate fatality during the sixth week of malignant diphtheria has made us very cautious in the use of gavage.

*Other Considerations.*—In the foregoing discussion symptomatic therapy was emphasized, but it should be pointed out that this treatment is indicated for the physiologic disturbances of diphtheritic intoxication. The treatment of shock is aimed chiefly at supporting the circulation by the administration of solutions of sodium chloride and of dextrose and by transfusions of blood. The treatment for hepatic necrosis is the administration of dextrose, which not only supports the carbohydrate metabolism of the whole body but leads to more rapid repair of the liver.[11] In the treatment for nephritis the fluid intake must be maintained in order to prevent dehydration from aggravating any tendency to anuria.

No mention has been made in this discussion of specific therapy with diphtheria antitoxin because we believe that its use is too well established to warrant further comment.

The use of insulin in conjunction with dextrose in the treatment of malignant diphtheria has aroused considerable interest. It has been recommended and employed by some investigators [12] on the basis of the clinical and experimental evidence of disturbed carbohydrate metabolism during malignant diphtheria, which has been attributed to a true deficiency of insulin or to pancreatic dysfunction.[12a] No clinical reports attempting to evaluate insulin and dextrose in comparison with dextrose alone in the treatment of severe diphtheria are available. In the experimental work reported in our earlier articles [2] all the changes found that were indicative of a disturbance in carbohydrate metabolism could

11. Richter, P. F.: Ueber Insulinbehandlung hepatargischen Zustandes, Med. Klin. **20**:1381, 1924. Herszky, P.: Zur Insulin-Traubenzucker-Therapie der Leberinsuffizienz, Klin. Wchnschr. **5**:1473, 1926. Knake, E.: Die Behandlung der Leberkrankungen mit Insulin und Traubenzucker unter Berücksichtigung des Kindesalters, Ztschr. f. Kinderh. **47**:502, 1929.

12. (*a*) Schwentker, F. F., and Noel, W. W.: Circulatory Failure of Diphtheria: I. Clinical Manifestations of Early and Late Failure, Bull. Johns Hopkins Hosp. **45**:276, 1929; II. Carbohydrate Metabolism in Diphtheria Intoxication, ibid. **46**:259, 1930; III. Treatment of Circulatory Failure of Diphtheria, ibid. **46**:359, 1930. (*b*) Benn.[4] (*c*) Kostyol.[4]

best be related to the derangement in hepatic function. Moreover, studies of the respiratory quotient during the experimental diphtheritic intoxication revealed no evidences of deficiency of insulin or of pancreatic dysfunction. We have, therefore, seen little reason for including insulin as a therapeutic agent in the treatment.

During the last five years we have had the opportunity of treating five patients with malignant diphtheria by means of some or all of the procedures herein outlined. Only two of these patients manifested symptoms sufficiently severe to present most of the problems discussed. With all the patients, however, the results were satisfactory and made us reasonably certain of the safety and of the value of the procedures involved. We present these suggestions as a safe basis for the development of a rational symptomatic therapy in clinics in which malignant diphtheria is common.

## REPORT OF CASES

The following case is described because it illustrates all the problems in a striking manner and because its severity proved a crucial test for the procedures previously described. Chart 1 is a graphic representation of the course and treatment.

CASE 1.—A girl, aged 5 years, was admitted to the New Haven Hospital on the third day of illness with extensive nasopharyngeal diphtheria. On admission the temperature was 37.8 C. (100 F.); the pulse rate, 110, and the respiratory rate 24 per minute. Bilateral cervical adenitis, a marked nasal purulent discharge and extensive tonsillar and peritonsillar edema were present. The diphtheritic membrane covered both tonsils and extended over the anterior pillars and lateral aspects of the soft palate. The heart was normal, and the liver was felt just below the right costal margin. The patient received an intramuscular injection of 60,000 units of antitoxin. An electrocardiogram made soon after admission showed no abnormalities of conduction. Because of the elevated figure for the nonprotein nitrogen of the blood (57 mg. per hundred cubic centimeters) a stormy course was anticipated. This proved to be the case. The patient vomited infrequently the first two days in the hospital, but on the fourth day the vomiting became more frequent, and it was necessary to stop all food and medication by mouth. At that time the nonprotein nitrogen was 143 mg. per hundred cubic centimeters; the liver had increased in size; the heart sounds had become faint; the electrocardiogram showed definite changes (chart 2*A*), and the blood pressure was 90 systolic and 50 diastolic. The subsequent course can best be seen from chart 1. The salient features were: persistence of the azotemia, marked enlargement of the liver, hypotension and cardiac dilatation with electrocardiographic evidence of severe myocardial damage which was first noted on the eighth and persisted until the eighteenth day (chart 2 *B* and *C*). The child exhibited marked cardiac failure from the tenth to the fifteenth day, and then improved rapidly. Palatal paralysis was noted on the sixteenth day, and weakness of the lower extremities with a loss of the deep reflexes, on the twentieth day. During the period of gravest illness, which was from the seventh to the twenty-ninth day, the patient received a total of 785 Gm. of dextrose parenterally, mostly intravenously. At the height of the cardiac failure, which was on the tenth day, she received a total of 1500 cc. of dextrose solution intravenously without untoward reaction. On the fifteenth day

digitalis was administered, and the subsequent improvement persisted until complete recovery took place. The patient received nothing by mouth for a period of eleven days, during which nausea and occasional retching were present. She was discharged from the hospital on the sixty-third day, at which time no cardiac abnormality could be detected. There were no sequelae, and when seen one year later the child was normal in every respect.

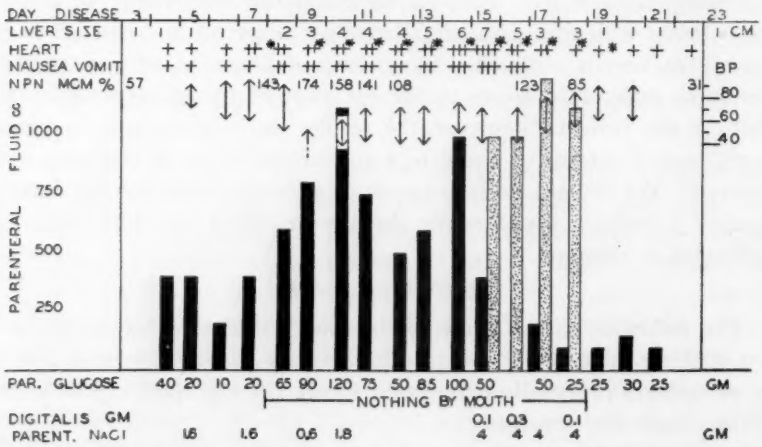


Chart 1 (case 1).—Graphic representation of the course of illness and treatment. The black areas indicate intravenous, and the stippled areas, subcutaneous, injections. The asterisks indicate a disturbance of conduction revealed by electrocardiograms.

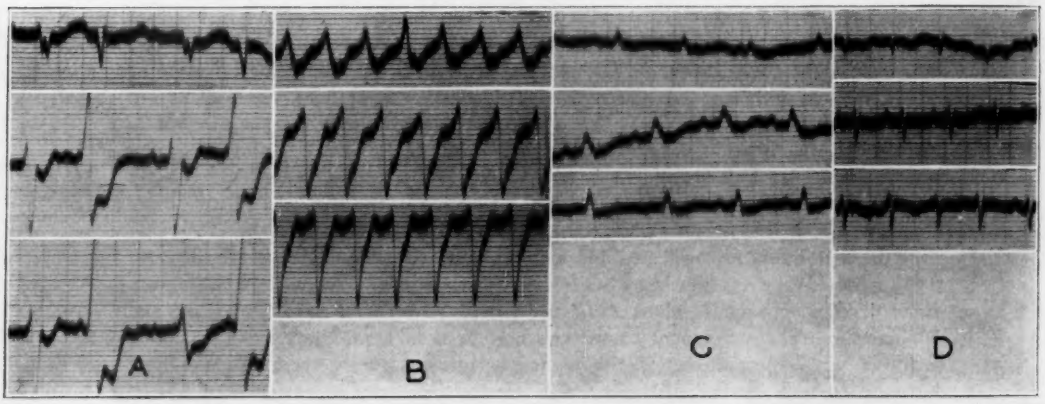


Chart 2 (case 1).—Electrocardiograms of the patient the course of whose illness is shown in chart 1. The leads are arranged in the standard order from above downward. *A* is a record made on the eighth day of illness: rate, 100 per minute; coupled rhythm; intraventricular heart block; partial auriculoventricular heart block with delayed conduction. *B* is a record made on the ninth day of illness: rate, 190 per minute; paroxysmal ventricular tachycardia. *C* is a record made on the seventeenth day of illness: rate, 110 per minute; intraventricular heart block; shifting pacemaker. *D* is a record made on the twenty-fourth day of illness: rate, 130 per minute; sino-auricular tachycardia.

Although in three of the remaining four cases of severe diphtheria the electrocardiograms showed changes comparable to those in this patient, in only one other case were the symptoms of cardiac failure marked enough to warrant the use of digitalis. In this patient, however, a change for the better took place soon after the first dose (0.1 Gm.) was administered. Because of this no more digitalis was given, and we did not believe that the drug played any decisive rôle in the subsequent recovery.

Four of the five children with malignant diphtheria recovered completely.

There was one fatal case.

CASE 2.—A boy, aged 8 years, came to the hospital on the fifth day of illness, with an extensive membrane covering both tonsils, marked cervical adenitis and periglandular edema. The child was quite toxic; the nonprotein nitrogen of the blood was 36 mg. per hundred cubic centimeters, but there was practically no vomiting. He was able to take moderate amounts of fluid with added dextrose by mouth, and was also given daily infusions of dextrose solutions. There was no evidence of cardiac involvement until the fourteenth day of the illness, when the blood pressure fell to 76 systolic and 60 diastolic and the electrocardiogram showed intraventricular heart block. With the therapy as described he did relatively well, and by the twenty-first day seemed well on the way to recovery. On the thirty-sixth day palatal paralysis was first noted, manifested by difficulty in swallowing and by nasal regurgitation. Oral feeding was reduced to a minimum, and the patient was sustained by parenteral administration of fluids. On the forty-first day there developed a rapidly spreading tonsillar membrane from which virulent diphtheria bacilli were subsequently recovered. The patient was desensitized and given 20,000 units of antitoxin intramuscularly. The membrane disappeared within two days, and except for the palatal paralysis the patient seemed to be doing well. On the forty-eighth day gavage was attempted because of the prolonged period of starvation to which he had been subjected. By fluoroscopic visualization it was found exceedingly simple to intubate the left bronchus, a fact which accounted for the difficulty experienced, before resorting to fluoroscopy, in guiding the tube into the stomach. The tube was allowed to remain in place for twelve hours, and two feedings were given. On the forty-ninth day the child suddenly became cyanotic and showed marked respiratory distress. An examination disclosed a shift of the mediastinum to the right with an absence of breath sounds on that side. The child died soon thereafter. At autopsy a complete atelectasis of the right lung and of the lower lobe of the left lung was found. The heart, microscopically, showed an acute diffuse myocarditis, and the liver showed extensive focal necrosis. The bronchi contained no excessive secretions or aspirated food.

## SUMMARY

A brief discussion of the therapy of malignant diphtheria is given. Means of combating vomiting, cardiac failure and the effects of starvation are presented. The parenteral administration of solutions of dextrose and sodium chloride and transfusions of blood seem to offer the best means of controlling the intractable vomiting and the physiologic

disturbances of shock and hepatic and renal insufficiency. Cardiac failure is not a contraindication for these procedures. Digitalis may be given safely in selected cases of cardiac decompensation if the effects are controlled by clinical and electrocardiographic observations. A report of a case, with a chart, illustrates the course of a severe case of diphtheria in which the procedures discussed were employed.

# IMMUNIZATION AGAINST WHOOPING COUGH

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Whooping cough is more often a serious disease of infancy and early childhood than is commonly believed. Recent epidemiologic studies by Sundal [1] of Stockholm and by Gundel and Schlüter [2] of Heidelberg, based on the last five annual epidemiologic reports of the League of Nations, show that it now causes death of more children under 5 years of age than does diphtheria, measles or scarlet fever. In children under 2, it causes more deaths than do these three diseases combined. The mortality of afflicted infants less than 1 year old may reach 15 per cent. The league's report shows that in 1932 more than 300,000 cases of the disease were reported in the United States. The average age at which it is contracted is about 4 years; the peak is often reached during the second or third year of life. About four-fifths of our annual 6,000 or more deaths from whooping cough occur among children less than 3 years of age. City, state and national reports show that whooping cough differs from other contagious diseases in that nearly all of the deaths occur before the third year of life. Whooping cough is relatively benign after infancy. Such epidemiologic data emphasize the urgency of attempts at eradication early in life and warrant efforts at immunization.

The Bacillus pertussis vaccine used over a period of five and a half years as an immunizing agent [3] differs from other vaccines used for pertussis. It is made every few months from recently isolated, hemolytic strains of the Bordet-Gengou bacillus. The medium contains about 20 per cent of human blood (freshly defibrinated). The forty-eight hour growth is scraped off and suspended in physiologic solution of sodium chloride, which contains 0.5 per cent phenol. After a week the pure, sterile vaccine is diluted to contain 10,000,000,000 bacilli per cubic centimeter.

About 650 nonimmune infants and young children have been given the vaccine months before any exposure has occurred. The total dosage after six months of age is 8 cc. The vaccine is administered as follows: one cubic centimeter is injected just under the skin in the deltoid region of

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Read before the Chicago Pediatric Society, Nov. 21, 1933.

1. Sundal, A.: Acta pædiat. **14**:104, 1932.
2. Gundel, M., and Schlüter, W.: Zentralbl. f. Bakt. (Abt. 1) **129**:461 (Oct. 6) 1933.
3. Sauer, L. W.: Immunization with Bacillus Pertussis Vaccine, J. A. M. A. **101**:1449 (Nov. 4) 1933.

each arm; one week later, 1.5 cc. is injected in the biceps region of each arm; one week later 1.5 cc. is injected in the triceps region of each arm. The youngest group of children to whom vaccine was given consisted of 150 infants less than 3 months of age in the Cradle (Evanston), each of whom received a total of only 6 cc. Since none of these infants has been exposed to date, it is not known whether infants so young will develop an active immunity from 6 cc. Their local and systemic reactions to the injections were negligible. The age of the main group of nonimmune children receiving injections (now over 500 in number) ranged from 6 months to 4 years, with an average age of 14 months. If the series from the Cradle is included the average age is 11 months. Taking all factors into consideration, the best age for immunization is probably between the seventh and tenth months. The skin should be cleansed with alcohol and the syringe and needle sterilized by heat.

*Whooping Cough: Distribution of Ages of Private Patients*

| Years | Toeplitz (378 Cases) per Cent | Sauer (400 Cases) per Cent |
|---|---|---|
| Under 1 | 18 | 8 |
| 1 | 21 | 11 |
| 2 | 13 | 15 |
| 3 | 14 | 12 |
| 4 | 9 | 10 |
| 5 (kindergarten age) | 9 | 19 |
| 6–15 | 16 | .. |
| 6 | .. | 10 |
| 7–10 | .. | 8 |
| 11–77 | .. | 7 |
| Total | 100 | 100 |

Reactions to the injections are chiefly local, although a transient rise in temperature may occur within from four to thirty-six hours after an injection. Reactions are due to the bacilli and their products (endotoxin and toxin). The cloudiness of the vaccine is due to the bacilli. The particulate nature of the suspension is a factor in the slow absorption. A transient local redness, tenderness and induration may be noted in some instances. No reaction has been severe enough, however, to necessitate the postponement of the subsequent injection. More than 3,500 injections have been given without the occurrence of infection at the site of injection. Each mother is told, just before and after the first injection and after the subsequent injections, that if a local reaction or tenderness develops she should not be concerned. The local reaction usually reaches a peak about twenty-four hours after the injection, after which time the tenderness rapidly subsides. At the time of the subsequent injection a small, circumscribed residual induration of the skin or a subcutaneous nodule may be palpable. The persistence of these small nodules is probably due to the slow absorption which results from the toxic action of the vaccine on the subcutaneous tissues. They com-

pletely disappear in the course of several weeks after the last injection. Reassurance by the physician at the time each injection is completed will allay unnecessary apprehension. When the vaccine is properly administered, no untoward effects should be noted. If a marked reaction should follow an injection, the subsequent dose should not be increased, but an extra, bilateral injection should be given one week after the third injection; or the subsequent bilateral injections should be given ten days apart. The vaccine contains no alien serum and will not sensitize; nor will it cause anaphylactic reactions, serum sickness or the Arthus phenomenon. As the reactions have not been considered severe enough to warrant any deviation from the original technic, no attempt has been made to detoxify the vaccine. Because of the danger of decreasing or destroying the antigenic content of the vaccine, it does not seem advisable to introduce new or variable factors at this time. That is something for the future.

The 32 children used as controls in twenty-five of the families contracted unquestionable pertussis. Thirty of the children receiving injections were thus intimately exposed to their brothers or sisters, but all of them escaped. Exposures occurred from four months to four and a half years after the injections. Not one of the 170 nonimmune children receiving injections, subsequently exposed at play, in the classroom, etc., has contracted the disease.

The active immunity conferred by the vaccine seems to depend on the potency, dosage and interval of time (at least four months) between injection and exposure. It is advisable to adhere to the established technic which has yielded protection. To maintain its full potency, i. e., to delay deterioration, the vaccine should be kept in a refrigerator until it is used by the physician. It is an immunizing agent (like typhoid vaccine, diphtheria toxoid or scarlet fever toxin). No claims are made for it as a therapeutic agent, nor when exposure has already occurred or threatens. To assure sufficient time for the immunization process to be completed before exposure is likely to occur, injections should be given during the nonepidemic period. This requires about four months.

# ELIMINATION OF MILK-BORNE DISEASE

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DURHAM, N. C.

A discussion of the subject of the elimination of milk-borne disease involves the answering of four questions: 1. What diseases are milk-borne? 2. How frequent are outbreaks of milk-borne disease? 3. What is being done to reduce the frequency of milk-borne disease? 4. How can the milk supply be made absolutely safe and still be within the means of the majority?

*What Diseases Are Milk-Borne?* Epidemics of the following diseases definitely have been proved to have originated from the drinking of milk: Brucella infection (undulant or Malta fever), diphtheria, foot and mouth disease, scarlet fever, septic or streptococcic sore throat, tuberculosis and typhoid and paratyphoid A and B fever. Bacillary dysentery, especially in children, also is a milk-borne disease, but the milk is contaminated more often in the home than in a dairy.

*How Frequent Are Outbreaks of Milk-Borne Disease?* In America from 1881 to 1927, 791 outbreaks of milk-borne diseases were reported,[1] but many others have occurred.[2] Since 1924, the U. S. Public Health Service has sent annual questionnaires about milk-borne diseases to all state health officers and to all health officers of cities with a population of over 10,000.[3] The answers are compiled in table 1. In addition, the U. S. Public Health Service since 1930 has been gathering information on the frequency of Brucella infection (undulant or Malta fever); in

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From the Department of Pediatrics, Duke University School of Medicine, and Duke Hospital.

1. Armstrong, C., and Parran, T., Jr.: Further Studies on the Importance of Milk and Milk Products as a Factor in the Causes of Outbreaks of Disease in the United States, Pub. Health Rep., supp. 62, 1927.

2. Wilkinson, E.: Milk-Borne Streptococci Infections, Brit. M. J. **2**:494, 1931. Johnston, M. M.; Brown, A.; Tisdall, F. F., and Fraser, D. T.: Intestinal Infections in Infants, Am. J. Dis. Child. **45**:1 (Jan.) 1933. Johnston, M. M.; Brown, A., and Kaake, M. J.: Intestinal Infections in Infants and in Children—1930 and 1931 Series, ibid. **45**:498, 1933. Williams, A. W., and Gurley, C. R.: Milk-Borne Septic Sore Throat and Scarlet Fever and Their Relation to Beta Hemolytic Streptococci, J. Bact. **23**:241, 1932. Archibald, R. E., and Feemster, R. F.: A Small Milk-Borne Outbreak of Septic Sore Throat, New England J. Med. **206**: 1252, 1932.

3. Frank, L. C.: The Public Health Service Milk Sanitation Program, Washington, D. C., Office of Milk Investigation, 1931.

1931, 1,545 cases were reported, and in 1932, 1,407.[3] Brucella infection has occurred also in England, Sweden, Denmark and Holland.[4] Today tuberculosis probably is not spread by milk nearly so frequently as in times past. Prior to 1910, 25 per cent of the cattle slaughtered in London were stated to be tuberculous; 16 per cent of ordinary market milk in New York City contained tubercle bacilli,[5] while the figures for Berlin [6] and London [7] were 28 per cent and 22 per cent, respectively. In 168 cases of tuberculosis among children in Edinburgh in 1916, 78.4 per cent among patients under 5 years of age, and 70.3 per cent among those between the ages of 5 and 16 years were caused by bovine bacilli.[8] In a series of cases of tuberculosis of the bones and joints studied in Edinburgh in 1912, 62 per cent were bovine in origin.[9] In New York, however, in 1917, only 12.5 per cent of 88 tuberculous children under 5

TABLE 1.—*Milk-Borne Outbreaks Reported by State and City Health Officers of the United States for the Years 1924-1932, Inclusive* [3]

| | 1924 | 1925 | 1926 | 1927 | 1928 | 1929 | 1930 | 1931 | 1932 | Total |
|---|---|---|---|---|---|---|---|---|---|---|
| Typhoid | 34 | 31 | 49 | 23 | 25 | 28 | 27 | 21 | 23 | 261 |
| Paratyphoid A | 0 | 1 | 2 | 0 | 0 | 0 | 0 | 1 | 0 | 4 |
| Paratyphoid B | 1 | 1 | 0 | 2 | 0 | 1 | 0 | 0 | 0 | 5 |
| Diphtheria | 1 | 1 | 2 | 2 | 2 | 0 | 0 | 1 | 0 | 9 |
| Septic sore throat | 1 | 6 | 6 | 0 | 3 | 8 | 9 | 8 | 3 | 44 |
| Scarlet fever | 5 | 4 | 5 | 5 | 8 | 11 | 2 | 1 | 6 | 47 |
| Miscellaneous | 2 | 0 | 4 | 4 | 3 | 2 | 6 | 2 | 1 | 24 |
| Total | 44 | 44 | 68 | 36 | 41 | 50 | 44 | 34 | 33 | 394 |

years of age had bovine infections.[10] At the present time, most of the cases of tuberculosis are human in origin. The former frequency of

4. Douglas, S. R.: Milk-Borne Diseases, Brit. M. J. **2**:198, 1932.

5. Hess, A. F.: The Subsequent Health of Children Who Drank Milk Containing Tubercle Bacilli, J. A. M. A. **56**:1322 (May 6) 1911.

6. Rabinowitsch-Kempner, L.: Types of Tubercle Bacilli in Human Tuberculosis, Am. Rev. Tuberc. **15**:225, 1927.

7. McFadyean, J.: What Is the Common Method of Infection in Tuberculosis? J. Comp. Path. & Therap. **23**:239, 1910; The Common Method of Infection in Human and Bovine Tuberculosis, J. Roy. Inst. Pub. Health **18**:705, 1910; Tuberculosis, J. Comp. Path. & Therap. **27**:218, 1914. Miller, J.: The Prevalence of Living Tubercle Bacilli in Edinburgh Milk, Edinburgh M. J. **12**:156, 1914. Gloyne, S. R.: The Presence of the Tubercle Bacillus in Milk, Tubercle **13**:443 and 488, 1932.

8. Wang, Chung Yik: Incidence of Bovine Tuberculosis in Man, J. Path. & Bact. **21**:131, 1917.

9. Fraser, J.: Relative Prevalence of Human and Bovine Type of Tubercle Bacilli in Bone and Joint Tuberculosis Occurring in Children, J. Exper. Med. **16**:432, 1912.

10. Park, W. H., and Krumwiede, C., Jr., et al.: The Relative Importance of the Bovine and Human Types of Tubercle Bacilli in the Different Forms of Human Tuberculosis, J. M. Research **25**:313, 1911.

bovine infection in the United States can be seen in table 2. However, it has been estimated that in England and Wales, 4,000 new cases of infection with bovine tubercle bacilli occur every year and about 2,000 deaths are reported annually[4] (table 3). An excellent review of the subject of milk-borne tuberculosis has been published recently.[11]

*What Is Being Done to Reduce the Frequency of Milk-Borne Disease?* During the past eight years, over 450 American municipalities, located in twenty-six states, have adopted the U. S. Public Health Service milk sanitation program,[3] which emphasizes that: 1. The public

TABLE 2.—*Distribution of Human and Bovine Tuberculosis in 543 American Children*[10]

| Lesions | Children Under 5 Years | | 5 to 16 Years | |
|---|---|---|---|---|
| | Human | Bovine | Human | Bovine |
| Pulmonary | 35 | 1 | 14 | 0 |
| Adenitis, axillary or inguinal | 2 | 0 | 4 | 0 |
| Adenitis, cervical | 15 | 24 | 36 | 22 |
| Abdominal | 10 | 14 | 8 | 9 |
| Generalized | 74 | 7 | 5 | 1 |
| Generalized, alimentary origin | 17 | 15 | 3 | 4 |
| Generalized and meningeal, alimentary origin | 5 | 10 | 1 | 0 |
| Generalized and meningeal | 76 | 1 | 10 | 0 |
| Meningeal | 28 | 4 | 3 | 0 |
| Bones and joints | 27 | 0 | 41 | 3 |
| Skin | 2 | 0 | 4 | 6 |
| Genito-urinary | 0 | 0 | 2 | 0 |
| Total | 291 | 76 | 131 | 45 |

TABLE 3.—*Human Lesions Due to Bovine Tubercle Bacilli (England)*[4]

| Lesion | Percentage Due to Bovine Tubercle Bacilli |
|---|---|
| Lupus | 51 |
| Cervical glands | 46 |
| Scrofuloderma | 31 |
| Meningitis (tuberculous) | 27 |
| Bone and joints | 18 |
| Genito-urinary | 17 |
| Pulmonary tuberculosis | 1 |

health supervision of milk supplies should be standardized voluntarily by the states, cities and counties in order to raise the average level of milk safety, to simplify the enforcement of milk sanitation by the health officer, to increase the consumption of milk, and to reduce the cost of milk sanitation. 2. This result may be achieved by the general adoption of the U. S. Public Health Service Milk Ordinance and Code.[12] 3. The pasteurization of all market milk is expedient and desirable in communities in which the predominant sentiment is favorable. In other com-

11. Pettersson, A.: The Transmission of Diseases by Means of Milk, Acta pædiat. **16**:286, 1933.

12. United States Public Health Service Milk Ordinance and Code, Washington, D. C., Office of Milk Investigation, 1931.

munities an adequate period of education of the consumer should precede any action on this matter by the local governing body. In all cases, the health officer should persistently recommend that all milk be properly produced and then properly pasteurized either in a commerial pasteurization plant or at home. 4. In order to maintain a high degree of excellence in local milk sanitation work, the state health department should rate the work of the local health departments at sufficiently frequent intervals. 5. In order that all milk-borne outbreaks may be recognized, an adequate epidemiologic method should be followed.

*How Can the Milk Supply Be Made Absolutely Safe, and Still Be Within the Means of the Majority?* In spite of the splendid results which have been obtained as a result of the program just outlined, many outbreaks of milk-borne disease still occur [13] (table 1). Furthermore, the expense of enforcing these provisions is so increasing the cost of milk that its consumption may become too expensive for the majority of the population. In addition, in spite of perfect inspection of the herd and the dairy, and in spite of efficient methods of distribution and of pasteurization, milk can become infected in the home through careless handling. A few dysentery bacilli on the mother's or nurse's hands or on flies' legs can produce fatal infections in infants. Furthermore, the contents of the duodenum and upper intestinal tract of infants dying of diarrhea are heavily infected with colon bacilli, a condition probably due to the diminution of pancreatic secretion caused by fever or hot weather, and the consequent accumulation of undigested and unabsorbed food. This food becomes infected with organisms that ascend from the colon and, by their growth, liberate irritating end-products.[14] Evidence also has been presented that saprophytic colon bacilli in cow's milk are harmful.[15] Therefore the use of lactic acid milk, which is bactericidal or antiseptic, should be encouraged. Buttermilk as an infant food was first recommended by Nils Rosen von Rosenstein [16] in 1776, but it soon fell into oblivion, and was not reintroduced in Holland until 1865,[17] in Germany until 1902,[18] in France until 1907 [19] and in the United States until

13. Armstrong and Parran.[1] Wilkinson et al.[2] Frank.[3] Douglas.[4]

14. Davison, W. C.: The Duodenal Contents of Infants in Health, and During and Following Diarrhea, Am. J. Dis. Child. **29**:743 (June) 1925.

15. Bessau, G.: Die Saprophyten der Milch und ihre Bedeutung, Acta pædiat. **16**:299, 1934.

16. Rosen von Rosenstein, N.: Diseases of Children and Their Remedies, London, Cadell, 1776, p. 62.

17. Ballot, A. M.: Karnemelk als Voedsel voor Kindern Beneden het Jaar, Nederl. tijdschr. v. geneesk. **2**:402, 1865.

18. De Mattos, T.: Die Buttermilch als Säuglingsnahrung, Jahrb. f. Kinderh. **55**:1, 1902.

19. Péhu, M.: Du babeurre dans les états pathologiques de la première enfance, Lyon méd. **109**:837, 1907. Marfan, A. B.: Le babeurre et le lait écrémé, Nourrisson **7**:321, 1919.

1919.[20] Gerstley said: "The popularity of buttermilk as an infant food is attested by the number of explanations offered for its success: sterility of milk, fine curd, low fat, lactic acid bacilli, effect of lactic acid on metabolism, gastric and intestinal asepsis, effect on peristalsis, effect of the food combination on metabolism, and combinations of these." [21] Rothey [22] showed that dysentery and typhoid bacilli inoculated into lactic acid milk are rapidly killed. An example of the reduction in infant death rate which follows the increased use of lactic acid milk and supervision is demonstrated by the report of Brooks [23] that the death rate of colored infants in a southern city decreased from 196 to 119 per thousand births, and of white infants from 83 to 57 during the four years in which the number of visits at infant feeding clinics increased from zero to 3,380. At these clinics, the mothers were instructed to feed their children lactic acid milk.

At present, whole lactic acid evaporated milk, which contains 7.5 per cent of added carbohydrate and 0.75 per cent lactic acid, probably is the best artificial infant food. A suitable preparation can be made by diluting unsweetened evaporated whole milk with an equal amount of

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20. Marriott, W. M.: The Artificial Feeding of Athreptic Infants, J. A. M. A. **73**:1173 (Oct. 18) 1919; M. Clin. North America **4**:717, 1920. Marriott, W. M.; Davidson, L. T., and Harshman, L. P.: Arch. Pediat. **39**:361, 1922. Marriott, W. M., and Davidson, L. T.: The Acidity of the Gastric Contents of Infants, Am. J. Dis. Child. **26**:542 (Dec.) 1923; Acidified Whole Milk as a Routine Infant Food, J. A. M. A. **81**:2007 (Dec. 15) 1923. Marriott, W. M.: Preparation of Lactic Acid Milk Mixtures for Infant Feeding, ibid. **89**:862 (Sept. 10) 1927. Marriott, W. M., and Schoenthal, L.: An Experimental Study of the Use of Unsweetened Evaporated Milk for the Preparation of Infant Feeding Formulas, Arch. Pediat. **46**:135, 1929.

21. Gerstley, J. R.: Infant Nutrition: VII. Lactic Acid Milk: Has It Solved the Problems of Infant Nutrition? Am. J. Dis. Child. **45**:538 (March) 1933. Saldun, M. L.: Le lait écrémé, acidifié avec l'acide lactique, Arch. de méd. d. enf. **34**:341, 1931. van Massenick, G.: Jahrb. f. Kinderh. **40**:756, 1904. Wurtz, A.: Med. Klin. **1**:1391, 1904. Rensburg, H.: Jahrb. f. Kinderh. **59**:74, 1904. Rummel, O.: Therap. d. Gegenw. **7**:259, 1905. Tada, G.: Monatschr. f. Kinderh. **4**:118, 1905. Dercherf, Elie: Arch. de méd. d. enf. **8**:1, 1905. Dunn, C. H.: Arch. Pediat. **24**:241, 1907. Klotz, M.: Jahrb. f. Kinderh. **70**:1, 1909. Menschikoff, M.: Monatschr. f. Kinderh. **9**:491, 1910. Stolte, K.: Jahrb. f. Kinderh. **74**:367, 1911; Monatschr. f. Kinderh. **11**:49, 1912. Langstein, L., and Meyer, L. F.: Säuglingsernährung und Säuglingsstoffwechsel, Munich, J. F. Bergmann, 1914. Ohta, H.: Jahrb. f. Kinderh. **85**:358, 1917. Leichtentritt, B.: ibid. **94**:119, 1921. Dohnal, E.: Monatschr. f. Kinderh. **27**:58, 1923. Hainiss, E.: ibid. **26**:568, 1923. Schiff, E.: Therap. d. Gegenw. **70**:90, 1929. Vogt, H.: Monatschr. f. Kinderh. **31**:30, 1925. Todd, T. W., and Kuenzel, W.: J. Lab. & Clin. Med. **15**:43, 1929. Engel, S.: Deutsche med. Wchnschr. **56**:433, 1930.

22. Rothey, K. B.: Is the Beneficial Effect of Lactic Acid Milk in Infant Feeding Due to the Bactericidal Action of the Lactic Acid? J. Pediat., to be published.

23. Brooks, B. U.: A Study of Infant Mortality in the Southern States, South. M. J. **23**:869, 1930.

cooled, boiled water, to which have been added previously 1.5 per cent of lactic acid (U. S. P.) and 15 per cent of cane sugar or molasses.[24] The home instructions are simple: The contents of a seven cent can of unsweetened evaporated milk (390 cc., 13 ounces) are poured into a quart bottle, previously scalded with boiling water. The empty can is nearly filled with boiling water, in which are then dissolved 1 teaspoonful of lactic acid (U. S. P.) and 3 level tablespoonfuls of cane sugar. As soon as this solution is cold, it is mixed slowly, while being stirred constantly, with the unsweetened evaporated milk. In order for the lactic acid to accomplish its bactericidal effect, the mixture should be kept for six hours before being fed in required amounts to infants. The final mixture (810 cc., 27 ounces) contains 100 calories per hundred cubic centimeters (30 per ounce), and is sufficient for a twenty-four-hour diet for any infant, regardless of age. Older children, of course, require solid food in addition. The mixture will remain sterile, even though not kept on ice. Whole lactic acid evaporated milk should never be repasteurized or boiled, as it will curdle. Cane sugar is a satisfactory carbohydrate, but molasses has the advantage of containing 0.0073 per cent iron and 0.27 per cent calcium. However, molasses sometimes may cause distention, loose stools or vomiting, especially in premature and weak infants. The caloric content of milk mixtures containing molasses or cane sugar will be approximately the same if both are measured in level teaspoonfuls, because the greater weight of the molasses compensates for its smaller number of calories. The reason that 7.5 per cent of carbohydrate is added to whole lactic acid evaporated milk is that if the amount is much less, the infants usually are constipated, and if more, their stools generally are loose. Furthermore, the caloric calculations are greatly simplified, for the resulting mixture contains one calory per cubic centimeter. The addition of carbohydrate to lactic acid milk is unnecessary for infants after the age of 8 months.

Infant foods should be judged by at least five criteria, all of which whole lactic acid evaporated milk satisfies: 1. Digestibility: Infants thrive on whole lactic acid evaporated milk at least as well as they do on breast milk.[25] The incidence of diarrhea, vomiting and bacillary dysentery is lower in infants fed lactic acid milk mixtures than in those receiv-

24. Davison, W. C.: The Compleat Pediatrician, Durham, N. C., Duke University Press, 1934.

25. Frank, L. C.; Clark, F. A.; Haskell, W. H.; Miller, M. M.; Moss, F. J., and Thomas, R. C.: Do Children Who Drink Raw Milk Thrive Better Than Children Who Drink Heated Milk? Pub. Health Rep. **47**:1951, 1932. Kositza, L.: A Comparative Study on the Use of Unsweetened Evaporated Milk and Bottled Cow's Milk in Infant Feeding, J. Pediat. **1**:426, 1932. Ross, J. B.: The Advantages of Evaporated Milk in Allergy and Infant Feeding, M. J. & Rec. **136**:276, 1932.

ing any other food, including breast milk. Marriott, who introduced the use of whole lactic acid evaporated milk,[20] and others have for several years advocated the use of this feeding and the author has used it for the past year. 2. Safety of source: The evaporation process employed in the preparation of the whole lactic acid evaporated milk removes the risk of Brucella infection (undulant or Malta fever), diphtheria, foot and mouth disease, scarlet fever, septic or streptococcic sore throat, tuberculosis, typhoid and paratyphoid A and B fever and other diseases which can be spread from a dairy. 3. Safety in home handling and in the infant's intestines: The demonstration by Rothey [22] that dysentery, typhoid and paratyphoid bacilli when added in large amounts to whole lactic acid evaporated milk are destroyed rapidly is an indication of a bactericidal power which no other infant feeding possesses. The antibacterial or lysozyme strength of breast milk is weak in comparison. Sweet milk mixtures even when boiled in the nursing bottle can be infected by the mother's hands while the nipple is being attached; whole lactic acid evaporated milk, because of its bactericidal activity, cannot readily be infected, either in the home or in the infant's duodenum and upper intestinal tract. 4. Cheapness: Whole lactic acid evaporated milk is cheaper than practically all other types of feeding; it costs only one half as much as the best grade of fresh milk, and three quarters as much as a fair grade. 5. Universal obtainability: Whole lactic acid evaporated milk is obtainable everywhere.

The only theoretical objections to the use of whole lactic acid evaporated milk are that the vitamins are destroyed, and that the taste is altered. However, all children between the ages of 2 weeks and 3 years, regardless of their type of feeding, should receive daily from 2 to 4 tablespoonfuls of tomato (or orange) juice to prevent scurvy, and from 2 to 4 teaspoonfuls of cod liver oil to prevent rickets. Some infants dislike whole lactic acid evaporated milk, especially those who previously have been fed with very sweet mixtures, but if the physician and mother cooperate, this difficulty can always be overcome. In fact, many of these children, after they have become accustomed to whole lactic acid evaporated milk, refuse to drink sweet milk—a consummation much to be desired. The addition of flavoring extracts, such as chocolate syrup, will improve the taste of the milk, especially for older children and adults.

## CONCLUSION

The incidence of milk-borne disease has been greatly diminished by the prophylactic measures which have been advocated up to the present, but outbreaks still occur, and, in addition, the expense of enforcing sanitary procedures has increased the cost of milk. The use of unsweetened evaporated milk insures a safe sterilized product, and if the bactericidal action of lactic acid is added, the risk of milk-borne disease is eliminated.

# STUDIES OF PHOSPHORUS OF BLOOD

## III. THE PHOSPHORUS PARTITION IN WHOLE BLOOD AND IN SERUM AND THE SERUM CALCIUM AND PLASMA PHOSPHATASE DURING HEALING OF LATE RICKETS

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The healing of late rickets has been observed in the pediatric clinic of the State University of Iowa coincident with the maintenance of a low level of inorganic phosphorus in the serum.[1] One possible explanation of this phenomenon is that sufficient ester phosphorus (acid-soluble organic phosphorus) was available at the site of the deposition of mineral so that, through the action of phosphatase, calcium phosphate could be deposited.[2] If this explanation is correct, an increase in the ester phosphorus of the blood without a concomitant increase in the inorganic phosphorus of the serum might result in healing of the rachitic process. This report presents the results of a study of the phosphorus partition in whole blood and in serum during healing of late rickets in two children. The serum calcium and the plasma phosphatase have also been studied. The methods used for phosphorus partition are discussed in detail in the first paper of this series.[3] Calcium was determined by the method of Kramer and Tisdall [4] and phosphatase according to Kay's technic.[5]

Three subjects have been studied. L. T., a girl aged 3 years, entered the hospital in April with severe rickets and marked deformities of the legs. The rickets proved very resistant to the usual methods of treatment, and healing was not complete until September. The second subject, E. E., a girl aged 12 years, had chronic acidosis of undetermined origin and active rickets of the low phosphorus type. Clinical improvement was observed after treatment with cod liver oil, sodium bicarbonate and a diet rich in calcium and phosphorus. For purposes of comparison, the results of examination of the blood of a child with healed rickets but persistently low values for serum inorganic phosphorus for three

From the Department of Pediatrics, College of Medicine, State University of Iowa.

1. Stearns, G., and Boyd, J. D.: J. Clin. Investigation **10**:591, 1931.
2. Robison, R.: Biochem. J. **17**:286, 1923.
3. Stearns, G., and Warweg, E.: J. Biol. Chem. **102**:749, 1933.
4. Kramer, B., and Tisdall, F. F.: J. Biol. Chem. **47**:475, 1921.
5. Kay, H. D.: J. Biol. Chem. **89**:235, 1930.

years have been included. The child was one of the two whose cases were previously reported [1] in whom rickets healed without an increase in serum inorganic phosphorus. Active rickets (roentgenologic evidence) was not present during the period when the inorganic phosphorus was consistently at a rachitic level.

The results of examination of the blood are given in tables 1 to 3 and charts 1 and 2. It will be noted that none of the children was

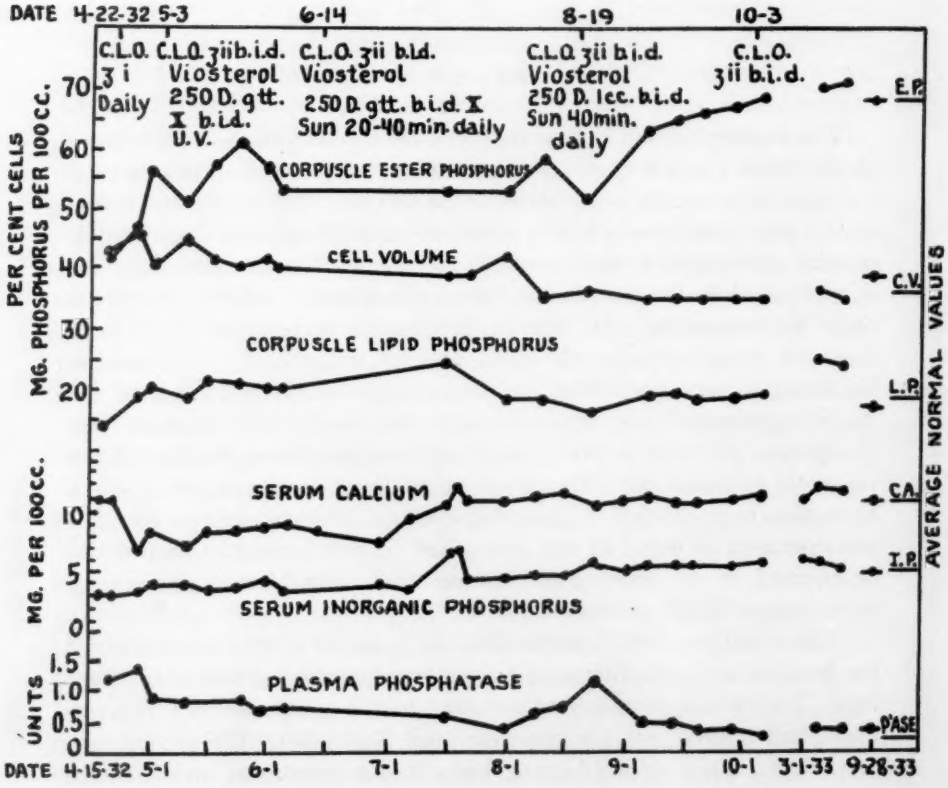


Chart 1.—Alterations in calcium, inorganic and ester phosphorus and phosphatase of blood during healing of late rickets in L. T., aged 3 years. Healing was complete according to roentgenologic evidence on Sept. 27, 1932.

anemic; in fact, the percentages of erythrocytes in the blood were above rather than below the average normal values for the given ages. This finding has been observed consistently in the children with late and resistant rickets studied in this clinic.

The values obtained for the youngest child, L. T., are illustrated in chart 1 and given in detail in table 1. On the patient's entrance to the hospital, the serum calcium was normal and the serum inorganic phos-

TABLE 1.—*Phosphorus Partition in Whole Blood, Corpuscles and Serum, Serum Calcium and Plasma Phosphatase of L. T.**

| Date | Corpuscles, per Cent | Whole Blood Phosphorus: Total | Whole Blood Phosphorus: Lipoid | Whole Blood Phosphorus: Inorganic | Whole Blood Phosphorus: Ester | Corpuscle Phosphorus: Lipoid | Corpuscle Phosphorus: Ester | Serum Phosphorus: Total | Serum Phosphorus: Lipoid | Serum Phosphorus: Inorganic | Serum Phosphorus: Ester | Serum Calcium, Mg. per 100 Cc. | Plasma Phosphatase, Units | Roentgenologic Report | Medication and Comment |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Av. normal 3-4 years | 36 | 41 | 12.0 | 4.3 | 23.5 | 18 | 66 | 14.0 | 8.6 | 5.1 | 0.5 | 10.8 | 0.21 | ..... | Stearns and Warweg [3] |
| 4/18/32 | .. | .... | .... | ... | .... | .... | .... | .... | ... | 3.3 | ... | .... | 10.8 | Evidence of advanced active rickets | |
| 4/20/32 | 43 | 31.8 | 10.8 | 2.9 | 17.9 | 14.2 | 41.5 | 11.3 | 8.2 | 3.2 | 0.1 | 10.7 | 1.2 | ..... | Cod liver oil, 1 tsp. daily, begun 4/22 |
| 4/27/32 | 46 | 38.1 | 13.7 | 3.1 | 21.3 | 18.9 | 46.2 | 13.3 | 9.4 | 3.5 | 0.3 | 6.6 | 1.4 | | |
| 5/ 2/32 | 40 | 39.1 | 13.7 | 2.4 | 22.8 | 20.2 | 56.1 | 13.7 | 9.3 | 4.0 | 0.6 | 7.8 | 0.86 | ..... | Cod liver oil, 2 tsp. daily; viosterol (250D), 20 drops daily; ultraviolet irradiations |
| 5/ 9/32 | 45 | 39.8 | 12.4 | 4.2 | 22.9 | 18.0 | 50.7 | 12.2 | 7.8 | 4.1 | 0.3 | 7.2 | 0.80 | | |
| 5/16/32 | 41 | 40.6 | 14.2 | 2.2 | 24.0 | 21.1 | 57.6 | 13.4 | 9.4 | 3.6 | 0.7 | 8.0 | .... | A little less irregularity of the epiphyseal margins | |
| 5/23/32 | 40 | 41.8 | 13.9 | 3.2 | 25.0 | 20.8 | 61.5 | 13.8 | 9.3 | 3.8 | 0.6 | 8.1 | 0.84 | | |
| 5/30/32 | 41 | 40.8 | 13.7 | 3.5 | 23.9 | 20.0 | 57.5 | 14.5 | 9.4 | 4.3 | 0.8 | 8.6 | 0.70 | ..... | Calcium monohydrogen phosphate, 1 tsp. daily, begun 5/27 |
| 6/ 4/32 | 40 | 38.3 | 13.0 | 3.7 | 21.7 | 20.2 | 53.1 | 12.5 | 8.2 | 3.4 | 0.8 | 8.7 | 0.71 | Definite and fairly marked improvement and healing | |
| 6/27/32 | .. | 41.3 | 13.9 | ... | .... | .... | .... | 14.3 | 8.6 | 3.7 | 0.3 | 7.1 | .... | ..... | Calcium monohydrogen phosphate discontinued 6/28 |
| 7/ 6/32 | .. | .... | .... | ... | .... | .... | .... | .... | ... | 3.3 | ... | 9.4 | | | |
| 7/16/32 | 38 | 42.0 | 15.3 | 6.1 | 20.3 | 24.6 | 53.4 | 16.4 | 9.6 | 6.7 | 0.0 | 10.5 | 0.59 | Slight gradual improvement continues; relatively no change in epiphyseal margin | Sunlight 40 minutes daily |
| 7/18/32 | .. | .... | .... | ... | .... | .... | .... | .... | ... | 7.0 | ... | 12.0 | | | |
| 7/25/32 | 38 | 41.0 | 13.7 | 3.0 | 24.6 | .... | .... | .... | ... | 4.4 | ... | 11.0 | | | |
| 8/ 1/32 | 42 | 39.3 | 12.3 | 4.0 | 22.6 | 18.3 | 58.1 | 13.3 | 8.0 | 4.6 | 0.5 | 11.0 | 0.45 | | |
| 8/ 8/32 | 35 | 36.9 | 12.2 | 3.7 | 20.7 | 18.1 | 58.0 | 14.7 | 9.0 | 4.8 | 0.3 | 11.3 | 0.63 | Calcium content subnormal; evidence of good reparative process | |
| 8/15/32 | .. | 40.6 | 12.5 | 4.3 | 23.9 | .... | .... | 17.0 | 10.4 | 4.8 | 1.6 | 11.5 | 0.68 | ..... | Viosterol (250D), increased to 2 cc. daily, 8/19 |
| 8/22/32 | 36 | 32.0 | 9.4 | 3.8 | 18.6 | 15.6 | 51.4 | 11.4 | 6.0 | 5.5 | 0.1 | 10.5 | 1.11 | | |
| 8/31/32 | .. | .... | .... | ... | .... | .... | .... | .... | ... | 4.9 | ... | 10.8 | .... | Some smoothing off of distal ends of radii and ulnas | |
| 9/ 6/32 | 35 | 40.5 | 12.7 | 4.9 | 22.9 | 19.1 | 64.5 | 14.8 | 9.2 | 5.2 | 0.4 | 11.3 | 0.50 | | |
| 9/13/32 | 35 | 40.8 | 13.3 | 4.4 | 23.2 | 20.0 | 65.1 | 14.4 | 8.9 | 5.2 | 0.6 | 10.8 | 0.51 | | |
| 9/19/32 | 35 | 41.0 | 12.5 | 4.6 | 23.4 | 18.1 | 66.0 | 14.9 | 9.5 | 5.1 | 0.4 | 10.7 | 0.43 | Definite marked improvement with increase in deposition throughout and smoothing out of epiphyseal margins | |
| 9/27/32 | 35 | 41.3 | 13.0 | 3.9 | 24.1 | 18.5 | 67.4 | 16.0 | 9.9 | 5.1 | 0.8 | 10.9 | 0.42 | Excellent improvement; no evidence of any active rickets present at this time | Viosterol discontinued; ultraviolet irradiation discontinued |
| 10/ 6/32 | 35 | 42.7 | 12.3 | 4.6 | 25.4 | 18.9 | 69.0 | 15.3 | 8.8 | 5.6 | 0.7 | 11.2 | 0.38 | | |
| 3/ 2/33 | .. | .... | .... | ... | .... | .... | .... | .... | ... | 6.0 | ... | 10.8 | .... | No roentgenologic evidence of rickets | |
| 3/ 5/33 | 36 | 44.0 | 14.7 | 3.9 | 25.5 | 23.4 | 70.0 | 16.2 | 10.1 | 5.7 | 0.5 | 11.6 | 0.48 | No roentgenologic evidence of rickets | |
| 3/11/33 | 35 | 43.7 | 14.3 | 4.1 | 25.2 | 23.1 | 71.4 | 15.3 | 9.6 | 5.1 | 0.4 | 11.5 | 0.44 | No roentgenologic evidence of rickets | |
| 9/27/33 | 38 | 37.2 | 11.7 | 4.0 | 23.8 | 16.7 | 67.6 | 13.8 | 8.5 | 5.2 | 0.2 | 10.8 | 0.41 | No roentgenologic evidence of rickets | |

* The phosphorus values are given in milligrams per hundred cubic centimeters.

phorus much reduced. Institution of the antirachitic treatment customary in the clinic resulted in a decided decrease in calcium without a corresponding increase in inorganic phosphorus, a finding often noted in infantile rickets during inadequate treatment. Moderate increases in the intake of vitamin D were without effect on the levels of calcium and inorganic phosphorus of the serum. Roentgenologic evidence of slow healing was observed.

During a period of very sunny weather in late June and early July, the whole body of the child was exposed to direct sunlight, the time of exposure being increased rapidly to forty minutes daily. The levels of serum calcium and serum inorganic phosphorus increased to normal during this period, but the level of phosphorus dropped again during a week of rainy weather. The dosage of viosterol (250 D) was then increased to 2 cc. daily, whereupon the inorganic phosphorus rose to, and remained consistently at, the average normal level. Complete healing (roentgenologic evidence) was observed five weeks after institution of the increased dosage of viosterol. In this child, then, healing was very slow, with one definite setback in midsummer.

The plasma phosphatase content at the time of admission was about five times the normal amount for a child of this age.[3] Smith and Maizels [6] have shown that in infantile rickets the plasma phosphatase is greatly increased and decreases as healing occurs, but does not reach normal until well after the serum inorganic phosphorus is normal and there is roentgenologic evidence that healing is complete. Apparently the plasma phosphatase follows this same course in late rickets. The value decreased rapidly at first, then more slowly through the period of conservative treatment. During the period of decrease in serum inorganic phosphorus in midsummer, the phosphatase increased again to almost its original value, decreasing as the phosphorus became normal. At the time of complete healing (roentgenologic evidence) the value for phosphatase was still from two to three times the normal figure. One year after healing was complete, it was still about twice the normal value, although no roentgenologic evidence of reactivity of the rickets was observed.

The changes in the ester phosphorus content of the erythrocytes were as marked in this child as were the changes in serum inorganic phosphorus. The value at entrance to the hospital was less than 60 per cent of the normal. Even with the conservative treatment at first given, the level of corpuscle ester phosphorus rose rapidly for a time. The increase could not be maintained, however, although the values remained above the low level observed at entrance. It appeared that a second rise in this component was attained with more difficulty than the early increase. The

6. Smith, J., and Maizels, M.: Arch. Dis. Childhood **7**:149, 1932.

level of serum inorganic phosphorus rose transiently in midsummer, but that of the ester phosphorus remained low. Only after the dosage of viosterol was increased to 2 cc. daily did the ester phosphorus attain the normal level, which was then maintained after discontinuation of the viosterol. The child was given 3 teaspoonfuls of cod liver oil (about 450 D) daily during the ensuing year, an amount apparently sufficient to maintain both the serum inorganic phosphorus and the corpuscle ester phosphorus at the normal levels.

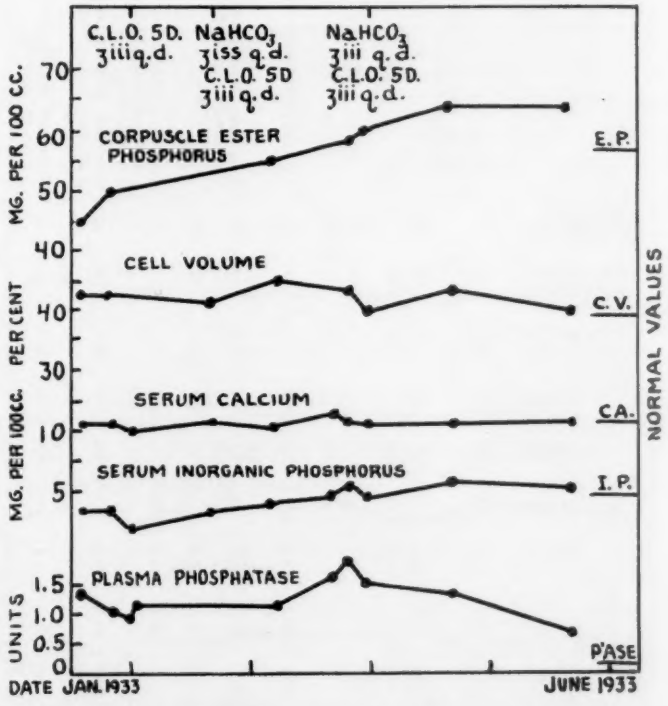


Chart 2.—Alterations in calcium, inorganic and ester phosphorus and phosphatase of blood during healing of late rickets in E. E., aged 12 years.

Roentgenologic studies showed evidence of very slow healing during the first two months of the child's stay in the hospital. During this time the inorganic phosphorus was at a low level, but the corpuscle ester phosphorus, while subnormal, was definitely above the level at entrance. The high level of the phosphatase of the plasma presumably indicates an increased phosphatase content of the bones. It seems possible that the amounts of ester phosphorus available, together with the inorganic phosphorus, were sufficient to allow some deposition of calcium phosphate during this period.

TABLE 2.—*Phosphorus Partition in Whole Blood, Corpuscles and Serum, Serum Calcium and Plasma Phosphatase of E. E.**

| Date | Corpuscles, per Cent | Whole Blood Phosphorus | | | | Corpuscle Phosphorus | | Serum Phosphorus | | | | Serum Calcium, Mg. per 100 Cc. | Plasma Phosphatase, Units | Carbon Dioxide, Vols. per Cent | Roentgenologic Report | Medication and Comment |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Total | Lipoid | Inorganic | Ester | Lipoid | Ester | Total | Lipoid | Inorganic | Ester | | | | | |
| Av. normal 12-15 yrs. | 38.6 | 39.7 | 11.6 | 4.2 | 21.8 | 16.1 | 56.2 | 13.8 | 8.6 | 4.7 | 0.6 | 10.7 | 0.18 | ... | ..... | Stearns and Warweg [3] |
| 1/20/33 | 43 | 33.8 | 11.0 | 2.6 | 19.9 | 15.2 | 45.1 | 12.0 | 8.0 | 3.0 | 0.8 | 10.6 | 1.43 | ... | Generalized decrease in calcium content; definite evidence of late rickets | Diet high in calcium; no vitamin D |
| 1/26/33 | 43 | 36.0 | 11.5 | 2.8 | 21.9 | 15.2 | 50.2 | 12.6 | 8.7 | 3.1 | 0.6 | 10.6 | 1.09 | 28 | | |
| 1/30/33 | .. | 32.2 | .... | 2.2 | 18.4 | .... | .... | .... | ... | 2.4 | ... | 10.3 | 0.92 | ... | ..... | Diet low in calcium, 1/29; cod liver oil (5D), 3 tsp. daily |
| 1/31/33 | .. | 32.0 | 12.0 | 2.8 | 17.5 | .... | .... | .... | ... | ... | ... | .... | 1.18 | 32 | | |
| 2/20/23 | 41 | 35.8 | 11.5 | 2.6 | 21.8 | .... | .... | .... | ... | 3.1 | ... | 11.0 | .... | 28 | ..... | Diet high in calcium, 2/1; medication as from 1/29 |
| 3/ 7/33 | 45 | 42.6 | 14.2 | 2.9 | 25.3 | 19.1 | 55.1 | 14.8 | 10.1 | 3.6 | 0.9 | 10.6 | 1.15 | 57.5 | Continued evidence of rickets | Sodium bicarbonate, 1½ tsp. daily |
| 3/22/33 | .. | .... | .... | ... | .... | .... | .... | .... | ... | 4.6 | ... | 11.5 | 1.63 | | | |
| 3/23/33 | 42 | 39.6 | 12.0 | 3.6 | 24.2 | 16.7 | 57.6 | 13.9 | 8.7 | 5.3 | 0.0 | 10.8 | 1.88 | 35† | Continued evidence of rickets; calcium content subnormal | |
| 3/29/33 | 40 | 39.5 | 11.6 | 3.9 | 24.2 | 17.0 | 60.0 | 12.7 | 8.0 | 4.5 | 0.4 | 10.5 | 1.61 | 36† | ..... | Sodium bicarbonate, 3 tsp. daily |
| 4/21/33 | 44 | 45.2 | 11.6 | 4.6 | 28.7 | 15.6 | 64.5 | 14.8 | 8.6 | 5.6 | 0.5 | 10.6 | 1.45 | 35† | Continued evidence of rickets; calcium content slightly subnormal | |
| 5/22/33 | 40 | 42.1 | 12.4 | 4.6 | 26.0 | 19.0 | 64.5 | 13.3 | 8.0 | 5.2 | 0.3 | 10.9 | 0.65 | 58‡ | Rachitic process apparently healing; calcium content normal | |
| 8/ 1/3 | .. | .... | .... | ... | .... | .... | .... | .... | ... | 5.5 | ... | 11.5 | .... | ... | Excellent healing and deposition of calcium | |

* The phosphorus values are given in milligrams per hundred cubic centimeters.

† Before the ingestion of sodium bicarbonate.

‡ After the ingestion of sodium bicarbonate.

The lipoid phosphorus of the corpuscles was at a low level at the time of first observation. The value increased rapidly to above the normal level, then fluctuated more or less during the following year. The alterations in this component of phosphorus bore no apparent relation, however, to the healing of the rickets.

The results of examination of the blood of the second patient (E. E., 12 years) are given in table 2 and chart 2. Preceding treatment, the values for serum inorganic and corpuscle ester phosphorus were similar to those noted in the case of the younger child, L. T., both being defi-

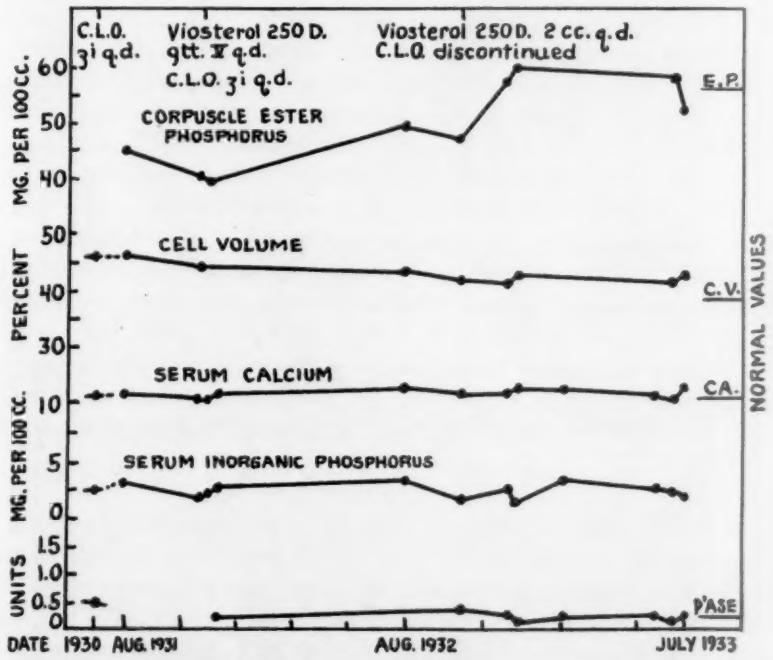


Chart 3.—Alterations in calcium, inorganic and ester phosphorus and phosphatase of blood of M. N. over a period of three years. Active rickets was not observed during this time, but growth was much retarded.

nitely low. The increase of corpuscle ester phosphorus, moreover, preceded that of inorganic phosphorus. In this patient, however, the value for ester phosphorus remained high during the course of healing. The value for plasma phosphatase, which was very high at first, decreased slowly, and was probably still well above normal when roentgenologic healing was complete.

The third subject, M. N. (table 3), was one of the two whose cases were previously reported.[1] No roentgenologic evidence of reactivity of rickets had been observed since 1930. At no time, however, had the

TABLE 3.—*Phosphorus Partition in Whole Blood, Corpuscles and Serum, Serum Calcium and Plasma Phosphatase of M. N.**

| Date | Age, Years | Corpuscles, per Cent | Whole Blood Phosphorus | | | | Corpuscle Phosphorus | | Serum Phosphorus | | | | Serum Calcium, Mg. per 100 Cc. | Plasma Phosphatase, Units | Roentgenologic Report | Medication and Comment |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Total | Lipoid | Inorganic | Ester | Lipoid | Ester | Total | Lipoid | Inorganic | Ester | | | | |
| **Av. normal** | | | | | | | | | | | | | | | | |
| 12-15 yrs. | .. | 38.6 | 39.7 | 11.6 | 4.2 | 22.0 | 16.1 | 56.2 | 13.8 | 8.6 | 4.7 | 0.6 | 10.8 | 0.18 | ..... | Stearns and Warweg [3] |
| 11/11/30 | 12 | .. | .... | .... | ... | 19.6† | .... | .... | .... | ... | 2.7 | ... | 10.9 | 0.45‡ | Healed rickets..... | Cod liver oil, 1 tbsp. daily |
| 8/17/31 | 13 | 46 | 36.3 | 12.0 | 2.7 | 21.4 | 14.2 | 45.9 | 13.5 | 10.1 | 2.8 | 0.5 | 11.3 | .... | No roentgenologic evidence of active rickets during period covered in this table | |
| 11/27/31 | 13 | 46 | 31.5 | 11.4 | 1.6 | 18.7 | 13.5 | 40.2 | 11.7 | 9.6 | 1.7 | 0.4 | 10.6 | .... | ..... | Viosterol (250D), 12 drops daily; cod liver oil, 1 tbsp. daily |
| 12/ 1/31 | 13 | 45 | 31.4 | 11.1 | 1.9 | 18.2 | 17.1 | 40.0 | 11.5 | 6.2 | 1.9 | 0.5 | 10.6 | | | |
| 12/ 5/31 | 13 | .. | .... | .... | ... | .... | .... | .... | 12.8 | 10.7 | 2.3 | 0.1 | 10.8 | 0.15 | | |
| 8/ 9/32 | 14 | 44 | 36.2 | 10.8 | 3.1 | 22.1 | 13.2 | 49.5 | 12.5 | 9.0 | 3.0 | 0.5 | 11.5 | | | |
| 10/16/32 | 14 | 43 | 35.6 | 12.8 | 1.6 | 21.0 | 17.4 | 47.2 | 12.8 | 9.4 | 1.9 | 1.2 | 11.2 | 0.37 | ..... | Viosterol (250D), 2 cc. daily; cod liver oil discontinued |
| 12/20/32 | 14 | 42 | 39.2 | 12.4 | 2.2 | 24.3 | 16.2 | 57.4 | 12.6 | 9.6 | 2.4 | 0.4 | 11.3 | 0.23 | | |
| 12/22/32 | 14 | 43 | 40.5 | 10.9 | 1.5 | 26.0 | 13.3 | 60.0 | 12.5 | 8.9 | 1.8 | 0.5 | 11.6 | 0.17 | | |
| 2/16/33 | 15 | .. | .... | .... | ... | .... | .... | .... | .... | ... | 3.2 | ... | 11.6 | 0.23 | | |
| 6/14/33 | 15 | .. | .... | .... | ... | .... | .... | .... | .... | ... | 2.5 | ... | 10.6 | 0.25 | | |
| 7/11/33 | 15 | 43 | 40.5 | 12.5 | 2.5 | 25.3 | 16.7 | 58.4 | 12.5 | 9.4 | 2.3 | 0.3 | 10.2 | 0.12 | | |
| 7/15/33 | 15 | 44 | 38.4 | 12.4 | 2.1 | 24.1 | 14.5 | 53.6 | 14.0 | 10.7 | 2.1 | 0.9 | 11.2 | 0.18 | | |

* The phosphorus values are given in milligrams per hundred cubic centimeters.

† Calculated from total acid-soluble phosphorus of whole blood and inorganic phosphorus of serum.

‡ Value on June 24, 1930.

value for serum inorganic phosphorus been the normal one for the patient's age, and values below 2 mg. per hundred cubic centimeters were not uncommon. The ester phosphorus concentration in the corpuscles was also definitely low. Both inorganic and ester phosphorus showed seasonal fluctuations, the values in the winter being markedly subnormal. The absence of active rickets during this time may possibly be explained by the slow growth of the child. At 12 years, her height was 135.9 cm. (53½ inches). Three years later she had grown only 3.8 cm. (1½ inches), a very slow rate of growth for this age period.

The plasma phosphatase, which had been high at the time of the first healing of the rickets (June 1930), had reached a normal level by December 1931. A year later (October 1932) an increased amount of phosphatase in the plasma was again observed, together with a decrease in the amount of inorganic phosphorus in the serum. From this time on, viosterol (250D) was given in a daily dosage of 2 cc. At the next observation (December 1932) the value for phosphatase was again normal, and a marked increase in the amount of corpuscle ester phosphorus was observed, notwithstanding the unfavorable season. The value for serum inorganic phosphorus was little changed in December, and reached the highest point two months later. Thus, in this subject as in the two with active rickets, a marked rise in the level of ester phosphorus was an early result of increased dosage of vitamin D, although the quantity of vitamin D given was apparently insufficient to maintain either ester or inorganic phosphorus at a normal level.

## COMMENT

The first determinations of phosphorus in the blood in rickets were those of Iversen and Lenstrup [7] in 1920. These authors reported that in infantile rickets the total values for acid-soluble phosphorus of whole blood and serum were much decreased, but rose to normal or above normal during healing. Cod liver oil was used as the antirachitic agent. The average values for total acid-soluble phosphorus in the whole blood and in the serum of four rachitic infants were 20.7 and 4.8 mg. per hundred cubic centimeters, respectively. After healing was complete, these average values had increased to 27.6 and 7.7 mg. per hundred cubic centimeters. The serum inorganic phosphorus was not determined but usually is about 0.5 mg. less than the serum acid-soluble phosphorus. The increase in the acid-soluble phosphorus of the whole blood is well above the amount which could have been contributed by an increase in inorganic phosphorus (about 3 mg.), there being an increase in ester phosphorus as well as in inorganic phosphorus during the healing process.

7. Iversen, P., and Lenstrup, E.: Foredr. v. nord. kong. f. paediat., 1920, p. 89.

The following year, Howland and Kramer [8] observed that a decrease in serum inorganic phosphorus was a constant finding in infantile rickets and that a rise in the level of this component was one of the first signs of healing. Zucker and Gutman [9] then studied the phosphorus partition in the blood in infantile rickets and reported that only the inorganic phosphorus was altered. These investigators, however, did not study the blood of the same infant before and after healing had occurred, but instead studied a group of nine rachitic infants from 8 to 17 months of age and compared the findings with those for a group of normal subjects from 1 month to 8 years of age. The wider age range of the control group, together with the fact that probably none of the infants received cod liver oil, possibly accounts for the low normal values observed. György [10] determined the total acid-soluble phosphorus (inorganic plus ester) in the whole blood in infantile rickets and found no apparent relation between the degree of rickets and the level of acid-soluble phosphorus of the whole blood. Stare and Elvehjem [11] found little or no lowering of the level of ester phosphorus of the corpuscles in the blood of rachitic calves, while, on the other hand, Kay [12] recently reported that in rachitic rats the acid-soluble phosphorus esters of the red blood cells may be from 25 to 35 per cent below the normal values and are increased toward the normal on the addition of vitamin D to the diet.

The evidence in the literature as to alterations in corpuscle ester phosphorus in rickets is thus conflicting. One reason for this apparent conflict may lie in the fact that the ester phosphorus fraction increases almost immediately with institution of treatment, even though the quantity of vitamin D ingested may be insufficient to maintain the improvement. The roentgenologic findings in the child L. T. indicate that slow healing may occur when the ester fraction only is increased. In infants with rickets, and in rachitic animals, healing is much more rapid than was observed in the children reported here, so that the rise in ester phosphorus might well be missed. It is also conceivable that a relatively small amount of sunlight might cause an increase in the level of ester phosphorus but be insufficient to increase the level of inorganic phosphorus appreciably in the blood of a rachitic infant.

The composition of the ester (organic acid-soluble) fraction of the blood phosphorus is not yet definitely established. It has been shown [13]

8. Howland, J., and Kramer, B.: Calcium and Phosphorus in the Serum in Relation to Rickets, Am. J. Dis. Child. **22**:105 (Aug.) 1921.

9. Zucker, T. F., and Gutman, M.: Proc. Soc. Exper. Biol. & Med. **20**:372, 1922-1923.

10. György, P.: Ztschr. f. Kinderh. **111**:210, 1926.

11. Stare, F. J., and Elvehjem, C. A.: J. Biol. Chem. **97**:511, 1932.

12. Kay, H. D.: J. Biol. Chem. **99**:85, 1932.

13. Kay, H. D., and Robison, R.: Biochem. J. **18**:755, 1924.

that only a part of the corpuscle ester phosphorus is apparently utilizable as substrate for the bone enzyme. It is possible that this fraction only of the ester phosphorus is affected by the vitamin D intake, and changes might easily be masked by coincident alterations in other forms of ester phosphorus.

A partial separation of the various forms of ester phosphorus on the basis of differing rates of hydrolysis has been attempted by various investigators.[14] Bakwin, Bodansky and Turner [15] have reported that the "readily hydrolysable fraction," which splits on boiling for ten minutes with normal acid, is decreased in infantile rickets. Determinations of this fraction were made on the blood of E. E. during the period of active rickets. The amounts found varied from 4.5 to 5.8 mg. per hundred cubic centimeters, values within the normal range for her age, as determined by Bomskov.[14]

In conclusion, the values reported here show that marked lowering of the ester phosphorus occurred in two children with untreated late rickets, and that definite increases in this fraction were the first alteration in the blood phosphorus on institution of treatment. Evidence of roentgenologic healing was observed when the ester phosphorus only was increased. On the other hand, it appears that a rise in the level of ester phosphorus does not always precede an increase in the level of serum inorganic phosphorus, for a transient rise in the level of inorganic phosphorus with maintenance of a lowered level of corpuscle ester phosphorus was observed in one of the children studied.

## SUMMARY

The serum calcium, the plasma phosphatase and the phosphorus partition in the whole blood and in the serum have been followed during the healing of late rickets in two children, and over a period of three years in a child whose rickets had healed without a corresponding rise in the level of inorganic phosphorus of the serum.

In active late rickets, the organic acid-soluble or ester fraction of phosphorus is definitely low, and rises rapidly on addition of vitamin D to the diet. The rise in the ester phosphorus of the corpuscles was observed before the rise in the inorganic phosphorus of the serum occurred. If the amount of vitamin D given is insufficient for rapid healing, the increased level of ester phosphorus may not be maintained.

The readily hydrolyzable (ten minute) fraction of the ester phosphorus was not significantly altered from the normal in one patient during the period of active rickets.

14. Bomskov, C.: Ztschr. f. physiol Chem. **210**:67, 1932.

15. Bakwin, H.: Bodansky, O., and Turner, R.: Proc. Soc. Exper. Biol. & Med. **29**:1238, 1931-1932.

The level of plasma phosphatase was markedly increased in the blood of both children with active rickets. The values tended to decrease as healing progressed, but were still far above normal when roentgenologic healing was complete. In one patient, the plasma phosphatase was still double the normal value a year after healing was complete.

Roentgenologic evidence of healing was observed in one child before any rise in the level of inorganic phosphorus occurred. The ester phosphorus at this time was still below the normal level, but well above the level noted at the first observation.

# USE OF PENTNUCLEOTIDE IN MEASLES AND IN PERTUSSIS

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In view of the demonstration by Jackson and others [1] of the beneficial effects of pentose nucleotides in agranulocytic angina and other toxic leukopenias, the action of pentnucleotide in measles and in pertussis, both of which ordinarily exhibit quantitative or qualitative abnormalities in the leukocyte count, seemed worthy of investigation.

White cell counts were made every day and differential counts every other day in sixty-three cases of measles in thirty-three of which pentnucleotide was administered, and in twenty-four cases of pertussis, in ten of which the same treatment was given. Uncomplicated early cases were selected as far as possible. Studies were begun in the cases of measles on the third day of the rash, at the latest, and in the cases of pertussis within the first five days of definite whooping. It was originally planned to administer 10 cc. of the drug intramuscularly on five consecutive days. However, this dose was reduced one-half after the first patient, a 2 year old girl with measles, had a very severe reaction on the second day of the rash about three hours after the injection. Another girl of 20 months, in the preeruptive stage, exhibited a similar, but much less severe, reaction after an injection of 5 cc. These reactions were characterized by cyanosis, chills, marked fever and convulsions.

In the cases of pertussis, five injections of 5 cc. each were administered in ten days. A slight chill followed the injection in one case.

Complications were slightly more frequent and more severe in the patients with measles and pertussis who were treated than in the control subjects (table 1). Two deaths from bronchopneumonia occurred among the patients with measles who were treated, one in the hospital and one shortly after the patient's discharge. In neither of these cases

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From the Pathological Laboratory of the Willard Parker Hospital, Department of Hospitals.

1. Jackson, H., Jr.; Parker, F., Jr.; Rinehart, J. F., and Taylor, F. H. L.: Studies of Diseases of the Lymphoid and Myeloid Tissues, J. A. M. A. **97**:1436 (Nov. 14) 1931.

TABLE 1.—*Complications in Cases of Measles and Pertussis*

| | Measles | | | | Pertussis | | | |
|---|---|---|---|---|---|---|---|---|
| | Treated Patients | | Control Patients | | Treated Patients | | Control Patients | |
| | Number | Per Cent | Number | Per Cent | Number | Per Cent | Number | Per Cent |
| Otitis | 5 | 15.1 | 6 | 20.0 | 1 | 10.0 | 3 | 21.4 |
| Mastoiditis | 1 | 3.0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Bronchopneumonia | 3 | 9.0 | 1 | 3.3 | 0 | 0 | 0 | 0 |
| Cervical adenitis | 0 | 0 | 2 | 6.6 | 0 | 0 | 0 | 0 |
| Reaction following injection | 2 | 6.0 | .. | ... | 1 | 10.0 | .. | .. |
| Death | 2 | 6.0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Total cases complicated | 11* | 33.3 | 8* | 26.6 | 2 | 20.0 | 3 | 21.4 |

* Several patients had more than one of the complications listed.

TABLE 2.—*Cell Counts in Cases of Measles*

| Day | Averages—All Cases | | | | Averages—Uncomplicated Cases | | | |
|---|---|---|---|---|---|---|---|---|
| | White Blood Cells | Granulocytes | Lymphocytes | Other Cells | White Blood Cells | Granulocytes | Lymphocytes | Other Cells |
| **Treated Patients** | | | | | | | | |
| 1 | 5,781 | 3,600 | 2,070 | 111 | 5,364 | 3,406 | 1,857 | 101 |
| 2 | 8,126 | 5,672 | 2,258 | 206 | 8,334 | 5,288 | 2,732 | 311 |
| 3 | 10,236 | 6,305 | 3,635 | 296 | 9,560 | 5,812 | 3,471 | 277 |
| 4 | 9,980 | 6,025 | 3,726 | 229 | 9,770 | 6,780 | 2,756 | 234 |
| 5 | 11,252 | 7,180 | 3,724 | 348 | 10,922 | 6,837 | 3,856 | 229 |
| 6 | 9,952 | 6,557 | 2,898 | 497 | 8,870 | 5,871 | 2,707 | 292 |
| 7 | 10,660 | 7,063 | 3,320 | 277 | 8,920 | 5,507 | 3,185 | 228 |
| 8 | 9,327 | 6,821 | 2,006 | 410 | 8,070 | 5,735 | 1,924 | 411 |
| 9 | 9,690 | 6,598 | 2,949 | 143 | 7,563 | 5,081 | 2,324 | 158 |
| 10 | 10,388 | 7,167 | 3,014 | 207 | 7,780 | 4,785 | 2,762 | 233 |
| **Control Patients** | | | | | | | | |
| 1 | 5,737 | 3,420 | 2,107 | 160 | 5,767 | 3,494 | 2,130 | 143 |
| 2 | 6,644 | 3,866 | 2,546 | 232 | 6,600 | 3,867 | 2,576 | 257 |
| 3 | 8,126 | 4,875 | 3,032 | 219 | 7,990 | 4,394 | 3,421 | 175 |
| 4 | 8,571 | 5,117 | 3,277 | 177 | 7,816 | 4,719 | 2,926 | 171 |
| 5 | 9,944 | 5,976 | 3,740 | 228 | 8,536 | 5,505 | 2,835 | 196 |
| 6 | 9,961 | 6,474 | 3,238 | 249 | 10,336 | 6,894 | 3,143 | 299 |
| 7 | 10,763 | 7,028 | 3,531 | 204 | 9,940 | 6,431 | 3,331 | 178 |
| 8 | 12,775 | 8,228 | 4,266 | 281 | 9,983 | 6,109 | 3,695 | 179 |
| 9 | 12,538 | 8,538 | 3,886 | 114 | 10,166 | 6,790 | 3,193 | 183 |
| 10 | 12,160 | 8,317 | 3,552 | 219 | 8,750 | 6,052 | 2,156 | 542 |

TABLE 3.—*Cell Counts in Cases of Pertussis*

| Day | Averages—All Cases | | | | Averages—Uncomplicated Cases | | | |
|---|---|---|---|---|---|---|---|---|
| | White Blood Cells | Granulocytes | Lymphocytes | Other Cells | White Blood Cells | Granulocytes | Lymphocytes | Other Cells |
| **Treated Patients** | | | | | | | | |
| 1 | 22,045 | 7,253 | 14.573 | 220 | 19,975 | 7,290 | 7,489 | 199 |
| 2 | 24,227 | ..... | ..... | ... | 22,128 | ..... | ..... | ... |
| 3 | 22,518 | 7,498 | 14,592 | 428 | 21,170 | 7,890 | 12,858 | 402 |
| 4 | 24,822 | ..... | ..... | ... | 23,345 | ..... | ..... | ... |
| 5 | 24,108 | 7,955 | 15,912 | 241 | 22,667 | 8,322 | 12,984 | 361 |
| 6 | 23,887 | ..... | ..... | ... | 21,952 | ..... | ..... | ... |
| 7 | 22,156 | 7,998 | 13,826 | 332 | 19,901 | 7,423 | 12,140 | 338 |
| 8 | 21,588 | ..... | ..... | ... | 19,641 | ..... | ..... | ... |
| 9 | 21,045 | 6,987 | 7,576 | 589 | 19,070 | 6,789 | 11,862 | 419 |
| 10 | 18,367 | ..... | ..... | ... | 17,493 | ..... | ..... | ... |
| 11 | 16,400 | 6,068 | 10,038 | 294 | 15,929 | 5,623 | 9,877 | 430 |
| 12 | 18,473 | ..... | ..... | ... | 17,201 | ..... | ..... | ... |
| 13 | 18,875 | 7,455 | 11,288 | 132 | 17,106 | 7,150 | 9,546 | 410 |
| 14 | 15,815 | ..... | ..... | ... | 13,457 | ..... | ..... | ... |
| **Control Patients** | | | | | | | | |
| 1 | 17,009 | 7,347 | 9,271 | 391 | 15,615 | 6,777 | 7,167 | 390 |
| 2 | 18,585 | ..... | ..... | ... | 16,601 | ..... | ..... | ... |
| 3 | 18,988 | 7,272 | 11,451 | 265 | 17,435 | 7,061 | 9,660 | 714 |
| 4 | 19,393 | ..... | ..... | ... | 17,937 | ..... | ..... | ... |
| 5 | 19,159 | 7,012 | 11,649 | 498 | 18,401 | 6,624 | 11,501 | 276 |
| 6 | 20,684 | ..... | ..... | ... | 19,736 | ..... | ..... | ... |
| 7 | 21,195 | 7,509 | 13,135 | 551 | 20,782 | 7,045 | 13,156 | 581 |
| 8 | 19,853 | ..... | ..... | ... | 18,247 | ..... | ..... | ... |
| 9 | 22,767 | 7,103 | 14,686 | 978 | 22,552 | 7,038 | 14,514 | 969 |
| 10 | 20,666 | ..... | ..... | ... | 19,461 | ..... | ..... | ... |
| 11 | 20,873 | 8,244 | 11,949 | 680 | 21,107 | 7,598 | 12,579 | 927 |
| 12 | 19,777 | ..... | ..... | ... | 20,059 | ..... | ..... | ... |
| 13 | 22,261 | 8,859 | 12,762 | 690 | 21,020 | 7,357 | 13,033 | 630 |
| 14 | 22,422 | ..... | ..... | ... | 23,924 | ..... | ..... | ... |

was there any reaction after the injections, and there is no evidence that the pentnucleotide played any part as a cause of death.

From the clinical point of view, in both measles and pertussis, neither the course nor the severity of the disease was in any way affected by the drug, and no change could be demonstrated in the number or in the proportional distribution of the various types of white blood cells.

## CONCLUSIONS

1. Pentnucleotide has no effect on the course of the disease or the white blood cells in measles and in pertussis.

2. Severe reactions are prone to follow intramuscular injections of 10 cc. of the drug in children.

# IODINE CONTENT OF AMERICAN COD LIVER OIL

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Among the chemical elements which are present in animal and vegetable life in such infinitesimal amounts as to require estimation by micromethods or ultramicromethods iodine is unique in that its essential rôle in normal animal life was the first to be recognized. In the eighty years which have passed since the first observation of Chatin [1] that the incidence of goiter is in inverse relation to the iodine content of the food and water of a region, a rapidly expanding wealth of information has been accumulated as to the amount of this element available through food and drink to the inhabitants of many parts of the earth. It appears to be definitely established that the supply of iodine in the environment of persons living in regions in which enlargement of the thyroid gland is of merely sporadic occurrence is much greater than the supply in regions in which such enlargement is so common as to be called endemic.

Nevertheless not all dietary items have been examined with sufficient detail to enable one to estimate the probable daily iodine intake of persons in any region. Remington and his co-workers [2] have reported that the iodine content of samples of the same agricultural product grown in the same general locality may vary as much as from 200 to 300 per cent. Products of the sea are much richer in iodine than those of the land, and the content of that element is undoubtedly more constant in sea water than in soil, but Coulson [3] has reported large variations in the iodine content of oysters from different localities on the coast of the United States. It is apparent that before one can arrive at an average value for the amount of iodine contained in any article of food the analysis of a number of samples is necessary. It is also desirable that such information be obtained for as great a variety as possible of the articles generally used as food and drink. Since cod

Technical assistance was given by Harry von Kolnitz.

From the Research Laboratories, E. L. Patch Company, Boston, and the Medical College of the State of South Carolina, Charleston, S. C.

1. Chatin, A.: Compt. rend. Acad. d. sc. **33**:519, 529 and 584, 1851.

2. Remington, R. E.; Culp, F. B., and von Kolnitz, H.: J. Am. Chem. Soc. **51**:2942, 1929.

3. Coulson, E. J.: U. S. Bur. Fisheries, 1933, mem. S-334.

liver oil is widely used, in goitrous and nongoitrous regions alike, its iodine content is of interest.

Von Fellenberg [4] found 5,100 and 5,620 parts of iodine per billion parts in two samples of cod liver oil. Lunde [5] reported the figure as 15,200 parts per billion parts for cold-cleared oil, as 14,200 parts per billion parts for crude medicinal oil and as 9,900 parts per billion parts for pale medicinal oil, all the products considered originating from Norway. In calling attention to the difference between his values and those of von Fellenberg, Lunde [6] expressed the opinion that the iodine content of fish is dependent on the location of the fishing grounds. In 1929 Remington [7] began his attempts to outline a diet that would be low enough in iodine to produce goiter in rats. Desiring to use cod liver oil as a source of fat-soluble vitamins he examined two samples

TABLE 1.—*Iodine Content of Cod Liver Oil and of Some Common Food Materials* *

| | | | |
|---|---|---|---|
| Cod liver oil | 10,500 | Onion | 222 |
| Cod liver oil | 10,400 | Carrot | 213 |
| Lettuce | 912 | Potato | 211 |
| Spinach | 692 | Green bean | 210 |
| Beet tops | 657 | Blueberry | 206 |
| Summer squash | 625 | Eggplant | 187 |
| Cucumber | 530 | Artichoke | 182 |
| Carrot tops | 433 | Beet | 182 |
| Watermelon | 402 | Strawberry | 181 |
| Turnip tops | 376 | Dewberry | 169 |
| Rape | 300 | Peach | 162 |
| Asparagus | 285 | Pimento | 150 |
| Kale | 248 | Pea | 136 |
| Turnip | 271 | Tomato | 112 |
| Cabbage | 263 | Sweet potato | 98 |
| Collards | 232 | Peanut | 67 |
| Mustard | 224 | Pear | 65 |
| Okra | 223 | Oats in hull | 20 |

* The figures represent the parts of iodine per billion parts of each substance analyzed (dry basis).

of medicinal grade and found, respectively, 10,500 and 10,400 parts per billion parts of cod liver oil. These results showed that cod liver oil contains significantly greater quantities of iodine than commonly used food materials. Table 1 gives a comparison of the iodine content of the aforementioned kinds of cod liver oil with the iodine content reported by Remington [8] for fruits and vegetables grown on soil rich in iodine in South Carolina. It is apparent from the results reported in table 1 that the amount of iodine in the samples of cod liver oil examined is great enough to contribute materially to the iodine intake of persons using cod liver oil. Accordingly, it seemed important to make analyses

4. von Fellenberg, T.: Mitt. a. d. Geb. d. Lebensmittelunt. u. Hyg. **15**:246, 1924.

5. Lunde, G.: Nord. med. tidskr. **1**:475, 1929.

6. Lunde, G.: Chem. Rev. **6**:57, 1929.

7. Remington, R. E.: Unpublished data.

8. Remington, R. E.: J. Chem. Educat. **7**:2590, 1930.

of a larger number of samples of American cod liver oils, the results of which are embodied in this report.

The oils which are classed as "crude medicinal" (not destearinized) are from the principal producing areas of the North Atlantic Coast—Massachusetts, the area of George's and Brown's Banks,[9] Maine, Nova Scotia, Quebec (Gaspe Peninsula) and Newfoundland. A more detailed description of the sources and manner of obtaining the oils is published elsewhere.[10]

Five cubic centimeters of the oil was absorbed on a 6 inch (15.24 cm.) no. 3 cotton dental roll. A segment of Visking sausage casing

TABLE 2.—*Iodine Content of Cod Liver Oil* *

| | Sample Number | First Analysis | Second Analysis | Average | |
|---|---|---|---|---|---|
| Massachusetts | 1405 | 4,230 | 4,250 | 4,240 | |
| | 1388 | 4,430 | 4,320 | 4,270 | |
| | 1447 | 6,150 | 6,200 | 6,180 | 4,930 |
| Vicinity of George's and Brown's Banks [9] | 1387 | 4,820 | 4,590 | 4,710 | |
| | 1279 | 5,720 | 5,500 | 5,610 | |
| | 1280 | 4,570 | 4,640 | 4,610 | |
| | 1278 | 6,200 | 6,630 | 6,420 | 5,340 |
| Maine | 1466 | 3,820 | 3,360 | 3,590 | 3,590 |
| Nova Scotia | 1398 | 10,180 | 11,020 | 10,600 | |
| | 1391 | 14,570 | 13,910 | 14,240 | |
| | 1199 | 14,530 14,700 | 15,600 | 14,940 | 13,260 |
| Gaspe Peninsula, Quebec | 1365 | 12,900 | 13,200 | 13,050 | |
| | 1362 | 11,400 | 11,310 | 11,350 | |
| | 1360 | 10,400 | 9,270 | 9,840 | |
| | 1361 | 10,200 | 11,500 | 10,850 | |
| | 1363 | 10,600 | 11,740 | 11,170 | 11,250 |
| Newfoundland | 1318 | 6,900 | 7,280 | 7,090 | |
| | 1368 | 10,200 | 10,130 | 10,170 | |
| | 1371 | 7,760 | 8,030 | 7,900 | |
| | 1367 | 7,920 | 8,640 | 8,280 | 8,360 |
| General average | | | | 8,460 ± 530 | |

* The figures represent the parts of iodine per billion parts of cod liver oil.

was slipped over the roll to prevent the oily cotton from coming in contact with the apparatus. Subsequently the roll was burned in the enclosed torch devised by von Kolnitz and Remington.[11] The iodine was extracted with carbon tetrachloride from the combined liquids of the absorption bottles and washings of the torch and estimated colorimetrically. All the analyses were made in duplicate, as shown in table 2.

The table shows some interesting relationships. 1. The range of values (from 3,590 to 14,940 parts of iodine per billion parts of the substance analyzed) coincides quite well with values previously reported by

9. That vicinity is considered to produce deep sea cod.

10. Holmes, A. D., and Remington, R. E.: Indust. & Engin. Chem. **26**:573, 1934.

11. von Kolnitz, H., and Remington, R. E.: Indust. & Engin. Chem. (Anal. Ed.) **5**:38 1933.

different analysts, and the average value for the twenty samples (8,640 ± 530 parts per billion parts) falls about midway between the results of von Fellenberg [4] and those of Lunde.[5]

2. The iodine content of the oil apparently varies with the locality in which it is produced, the average values for the different localities being: Nova Scotia, 13,260 parts of iodine per billion parts of oil; Gaspe Peninsula, 11,250; Newfoundland, 8,360; area of George's and Brown's Banks, 5,340; Massachusetts, 4,930, and Maine, 3,590.

The data at present available are inadequate to explain the difference in the iodine content of oils produced along the shores of Maine and Massachusetts and that of oils produced farther north. It should be remembered that cod liver oil is produced on the shores of Massachusetts during the winter and in the northern areas during the summer. A careful study of the life habits [12] of the codfish reveals that it is a migratory fish and indulges in extensive migrations; thermal migrations are caused by a preference for water of from 35 to 50 F.; feeding migrations result from the search for food, and breeding migrations, which annually interrupt both the thermal and the feeding migrations, are caused by a search for shoal water in which to spawn. Hence, it is possible that oil is made from the same school of fish at northern points during the summer and at southern points during the winter.

The iodine content of sea water was first determined by Winkler,[13] who found 38 gammas per liter in the water of the Adriatic Sea. Since that time analyses of sea water have been made [14] at a number of places, and the value has been shown to be quite constant at from 18 to 23 gammas per liter, although this may be increased locally at times owing to the liberation of iodine from decaying vegetation. Hence it seems highly improbable that the differences reported for cod liver oil are influenced by the iodine which the codfish derives directly from sea water.

Obviously the iodine content of the food [15] of cod, which is a "ground fish," is highly significant in considering the source of iodine in cod liver oil. Lunde [6] reported that the amount of iodine in the flesh and intestinal content of the cod, which feeds mainly on the invertebrates found at the bottom of the ocean, is much higher than that in surface fish such as mackerel, herring, etc. Also Tressler and Wells [16] found that crustaceans and shell-fish are richer in iodine than vertebrate

12. Bigelow, H. B., and Welsh, W. W.: U. S. Bur. Fisheries, Bull. 40, 1925, pt. 1.

13. Winkler, L. W.: Ztschr. f. ang. Chem. **28**:447, 1915; **29**:68, 1916.

14. Orr, J. B., and Leitch, I.: Medical Research Council, Special Report Series, no. 123, London, His Majesty's Stationery Office, 1929.

15. Holmes.[10] Bigelow.[12]

16. Tressler, D. K., and Wells, A. W.: U. S. Bur. Fisheries, 1924, doc. 967.

fishes. It may be that these forms are more numerous in certain areas than in others and that this difference causes a variation in the iodine content of cod liver oil from different localities.

## COMMENT

Cod liver oil has been used for generations for the prevention and cure of various types of human disease. Doubtless this use resulted at first from empirical observations of the beneficial effects produced in certain disorders. Through the many decades during which it has been used many theories have been developed to explain its value, and as late as 1906 Cushny [17] did not believe that it had any virtue other than that of an easily digested fat. Since the development of the vitamin theory and the discovery that cod liver oil is one of the richest natural sources of vitamins A and D it has been assumed that the nutritional and therapeutic value of cod liver oil is due to its high vitamin content. Following the brilliant investigations of Hess,[18] Steenbock,[19] Rosenheim and Webster,[20] Windaus,[21] Pohl,[22] Bills and his co-workers [23] and others, who demonstrated that viosterol possesses unusual antirachitic activity, and the investigations of Steenbock,[24] Collison, Hume, Smedley-MacLean and Smith,[25] Moore,[26] von Euler, von Euler and Karrer [27] and others, who showed that carotene is a precursor of vitamin A, it was questioned whether these products could replace cod liver oil and other natural products as sources of vitamins A and D. However, several investigators have observed that the results obtained by administering carotene or viosterol do not parallel those obtained with cod liver oil when administered in amounts sufficient to supply equivalent vitamin units. For instance, Clausen [28] stated: "When rapid stor-

17. Cushny, A. R.: Textbook of Pharmacology and Therapeutics, ed. 4, Philadelphia, Lea Bros. & Co., 1906, p. 722.

18. Hess, A. F.: Experiments on the Action of Light in Relation to Rickets, Am. J. Dis. Child. **28**:517 (Oct.) 1924.

19. Steenbock, H.: Science **60**:224, 1924.

20. Rosenheim, O., and Webster, T. A.: J. Soc. Chem. Indust. **45**:923, 1926; Biochem. J. **20**:537, 1926.

21. Windaus, A.: Chem. Ztg. **51**:113, 1927.

22. Pohl, R.: Nachr. v. d. k. Gesellsch. d. Wissensch. z. Götting. Math-Phys. Klasse **2**:185, 1927.

23. Bills, C. E.; Honeywell, E. M., and Cox, W. M., Jr.: Biol. Chem. **80**:57, 1928.

24. Steenbock, H.: Science **50**:352, 1919.

25. Collison, D. L.; Hume, E. M.; Smedley-MacLean, I., and Smith, H. H.: Biochem. J. **23**:634, 1929.

26. Moore, T.: Lancet **2**:380, 1929.

27. von Euler, B.; von Euler, H., and Karrer, P.: Helvetica chim. acta **12**: 278, 1929.

28. Clausen, S. W.: Limits of the Anti-Infective Value of Provitamine A (Carotene), J. A. M. A. **101**:1384 (Oct. 28) 1933.

age of vitamin A is desired, halibut liver oil or cod liver oil would seem more suitable than preparations of carotene, because of the more rapid absorption of vitamin A than of carotene. Carotene is poorly absorbed in the presence of fever or diarrhea." Steenbock and his associates [29] stated that "the vitamin D produced by ordinary irradiation of ergosterol with a quartz mercury vapor lamp is a different substance from that found in cod liver oil."

As a consequence of such observations one questions whether the vitamins are really different, or whether the "plus" value of cod liver oil is due to other substances which, acting either independently or in conjunction with the vitamins, account for the favorable results produced.

According to Drummond and Hilditch,[30] Hupfer de l'Orme detected the presence of iodine in cod liver oil in 1836 and attributed its therapeutic properties to this constituent. Subsequently Andres,[31] Stanford,[31] Heyerdahl,[31] von Fellenberg [32] and others have shown that cod liver oil

TABLE 3.—*Absorption and Retention of Iodine from Various Sources* [34]

| | Percentage Absorbed | Percentage Retained |
|---|---|---|
| Potassium iodide | 89.5 | 34.4 |
| Cod liver oil | 93.4 | 47.4 |
| Sardines | 83.1 | 24.5 |
| Watercress | 64.8 | 12.9 |
| Sardines with oil removed | 88.2 | 9.5 |

contains significant amounts of iodine. That the iodine of cod liver oil is absorbed and passes into the milk of lactating animals has been shown by McClendon and his associates [33] in experiments on cows. Von Fellenberg [34] found a better retention of iodine from cod liver oil than from iodides, sardines, watercress or sardines with the oil removed. His data are given in table 3. The observations of von Fellenberg may be open to question since he considered the urinary elimination only, for Cole and Curtis [35] have shown that in women the urinary excretion

29. Steenbock, H.; Kletzien, S. W. F., and Halpin, J. G.: J. Biol. Chem. **97**:249, 1932.

30. Drummond, J. C., and Hilditch, T. P.: The Relative Value of Cod Liver Oils from Various Sources, London, His Majesty's Stationery Office, 1930, p. 8.

31. Cited by Lewkowitsch, J.: Chemical Technology and Analysis of Oils, Fats and Waxes, New York, The Macmillan Company, 1922, vol. 2, p. 448.

32. von Fellenberg, T.: Mitt. a. d. Geb. d. Lebensmittelunt. u. Hyg. **14**:161, 1923.

33. McClendon, J. F.; Remington, R. E., and von Kolnitz, H.: J. Am. Chem. Soc. **52**:541, 1930.

34. von Fellenberg, T.: Biochem. Ztschr. **142**:246, 1923.

35. Cole, V. V., and Curtis, G. M.: Proc. Soc. Exper. Biol. & Med. **31**:29, 1933.

of iodine is higher during the premenstrual and early menstrual periods than at other times, and Curtis and Philips [36] have reported that only a fraction of the iodine ingested is eliminated in the urine. Furthermore, there is a definite loss of iodine in sweat.[37] Curtis [38] has also found that the iodine of the blood is increased to about the same degree by the use of cod liver oil and of iodized salt.

On the basis of the aforementioned determinations of iodine the recommended [39] daily consumption of cod liver oil would supply 75 gammas of iodine. The minimal amount of iodine necessary to protect human beings against goiter, as pointed out by Levine and Remington,[40] is probably dependent on a number of factors, some of which are not clearly known. The work of von Fellenberg,[41] Lunde,[5] Bodnar,[42] Adolph [43] and Mazzocco [44] on the iodine intake of persons in goitrous and nongoitrous regions places the daily human requirement somewhere between 50 and 100 gammas. Hence the iodine content of 10 cc. of cod liver oil per day, if assimilated and made available for physiologic needs, would contribute materially to the requirements of the average human adult.

SUMMARY

The iodine content of twenty representative samples of American cod liver oil has been found to range from 3,590 to 14,940 parts per billion parts of cod liver oil, the average being 8,460 ± 530. Oils produced in the vicinity of Nova Scotia, Gaspe Peninsula and Newfoundland contained more iodine than those from Maine, Massachusetts or a region in which deep sea fish were available. The iodine content of daily doses of 10 cc. of these cod liver oils, if completely assimilated, would largely supply the requirements of the average human adult.

36. Curtis, G. M., and Philips, F. J.: The Urinary Excretion of Iodine in Toxic Goiter, J. A. M. A. **101**:1992 (Dec. 16) 1933; Ohio State M. J. **30**:149, 1934.

37. Scheffer, L.: Biochem. Ztschr. **259**:11, 1933.

38. Curtis, G. M.: Personal communication to the authors.

39. Pharmacopoeia of the United States, tenth revision, Philadelphia, J. B. Lippincott Company, 1926, p. 264.

40. Levine, H., and Remington, R. E.: J. Chem. Educat. **10**:649, 1933.

41. von Fellenberg, T.: Biochem. Ztschr. **152**:141, 1924.

42. Bodnar, J., and Straub, J.: Biochem. Ztschr. **227**:237, 1930.

43. Adolph, W. H., and Ch'en, S. C.: Chinese J. Physiol. **4**:437, 1930.

44. Mazzocco, P.: Semana méd. **37**:358, 1930.

# HISTOLOGY OF THE MIDOLIVARY REGION OF THE MEDULLA OBLONGATA IN THE NEW-BORN INFANT

## OBSERVATIONS ON VESSELS AND CELLS

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In a detailed study of the medullae of fifty-three still-born premature and full term children which we [1] made to determine the presence or absence of microscopic hemorrhages, certain observations of histologic interest repeated themselves. It seemed striking that there were so few vessels in the medulla (fig. 1), considering the importance of the structure, and not only how few but how small the largest of the vessels were. In measuring the thickness of the walls of 750 vessels it was found that they varied from the width of a single endothelial cell in the capillaries to 250 microns. The largest were found in the pia (still of cerebral type), where unexpectedly the arterioles exceeded the veins in number. This recalls Craigie's [2] work, except that he measured the diameters of capillaries instead of the thickness of their walls, so that he had data on the caliber, which, as he noted, differs in different parts of the brain and cord of the rat; he stated that corrections may be made for shrinkage. His figures show that the cerebellar cortex has a less rich supply than the other regions observed. If the physiology of the medulla were limited to the functions of the nuclei which have their origin in certain cranial nerves only, the amount of nourishment would seem scarcely adequate, but this is especially remarkable when one lists the centers attributed to it.

If human beings are "bags of sea water stuffed with cells," as Councilman said, perhaps the somewhat fluid appearance of the cells in these young subjects is accounted for; this is particularly notable in the cells lining the fourth ventricle and the ependyma and in those lying underneath the ependyma (fig. 2). Five hundred and thirty ependymal cells measured averaged 20 microns in length, and the

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1. Hemsath, F. A., and Canavan, M. D.: Am. J. Obst. & Gynec. **23**:471 (April) 1932.

2. Craigie, E. H., Jr.: J. Comp. Neurol. **53**:309, 1931.

cells were brilliantly colored. In figure 3 an ependymal wall, also from the midolivary region, but in an adult, shows how much smaller are the cells and how clear is the space below them, for in this location the ependymal wall is devoid of cells as far as the vascular bed beneath. Another examination of figure 2 shows gaps lateral to the ependymal cells, which leave distorted cells in the line but the ventricular

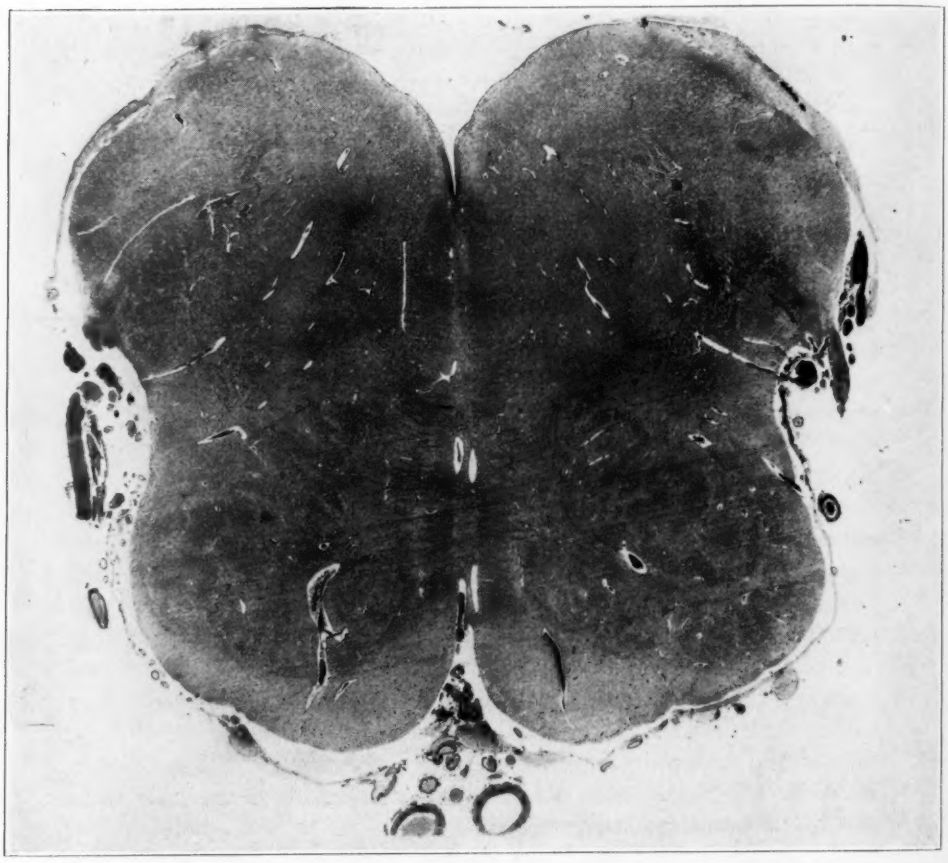

Fig. 1.—Cross-section of the medulla at the midolivary level, showing the thickness of the vessel walls in the pial membrane and in the substance of the medulla. Connective tissue stain; × 9.

edge intact, though by a most delicate stretch of protoplasm. Was this vacuole formed by a minute droplet of fluid which oozed through the membrane, or was it left as a space by the migration of some of these closely packed cells toward the blood vessels which nowhere reach to the ependymal line but form a bed below it? The presence of cells in this region, as shown in figure 2, suggests this second possibility.

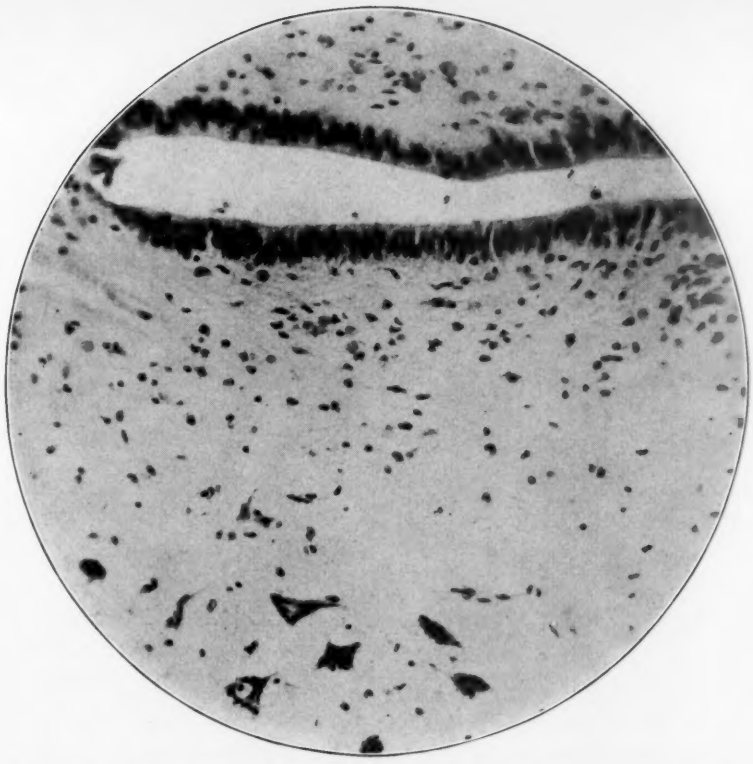

Fig. 2.—Section of the midolivary region of the medulla, showing the ependyma of a new-born infant. Note the size of the cells and the spaces between and the numbers of glia cells beneath; compare with figure 3. Cresyl violet stain; × 215.

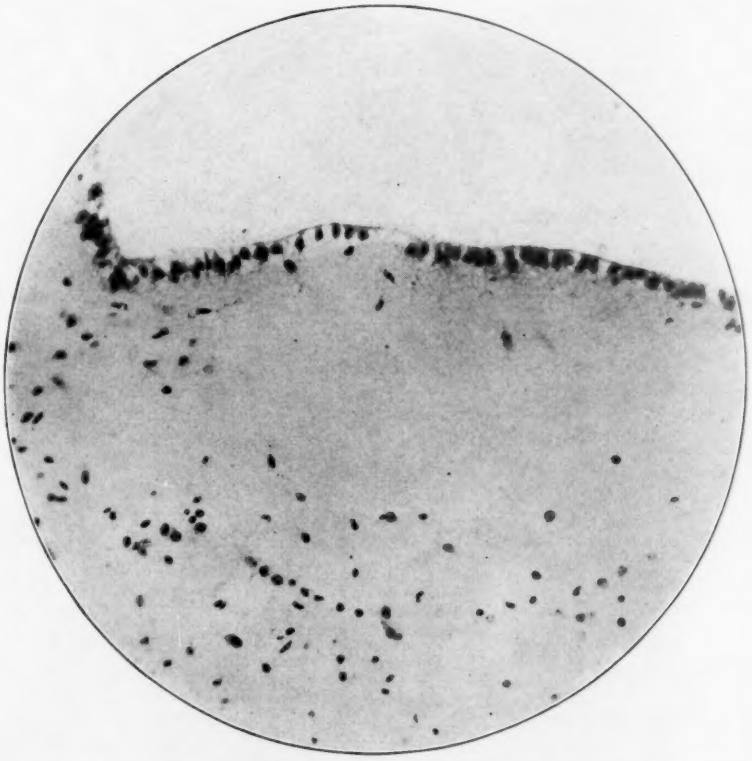

Fig. 3.—Section of the midolivary region of the medulla, showing the ependyma of an adult. Note the size of the cells and the almost complete absence of subependymal glia; compare with figure 2. Cresyl violet stain; × 215.

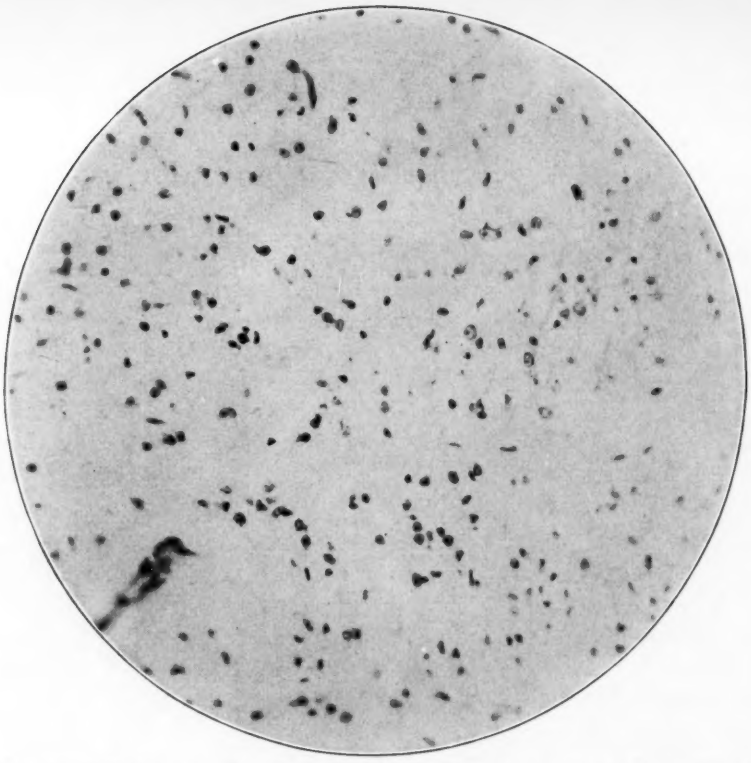

Fig. 4.—Cells between the olivary bodies, showing the types of glia: macroglia, microglia and oligodendroglia (the last stained brilliantly). Cresyl violet stain; × 215.

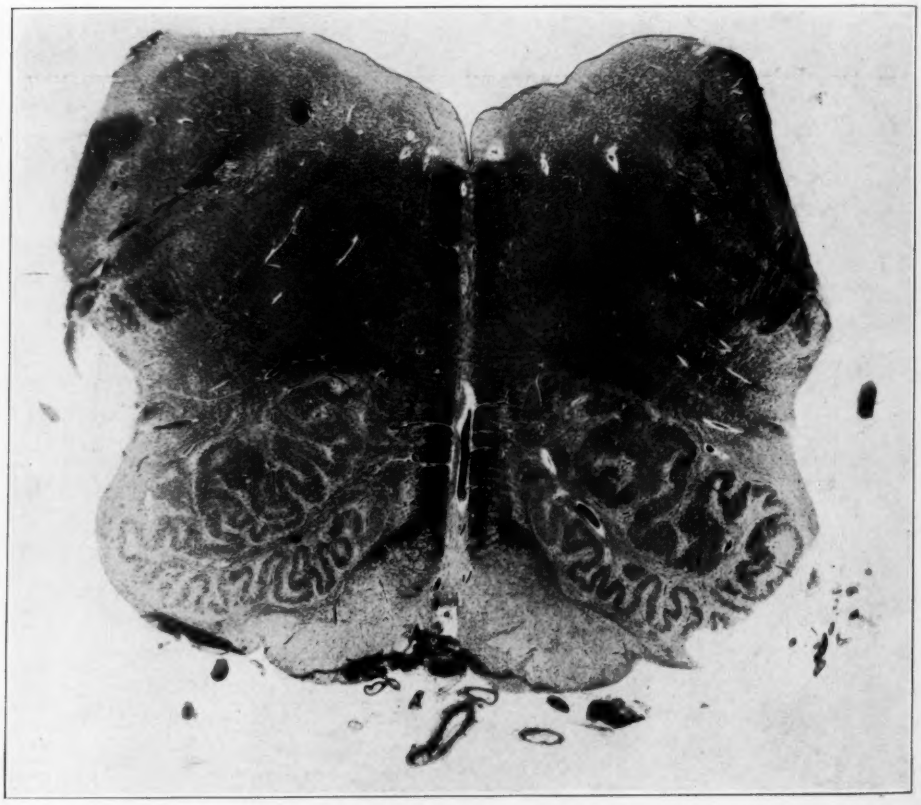

Fig. 5.—Cross-section of the midolivary region of the medulla of the same patient as the section seen in figure 4, showing small amounts of myelin. Weigert stain; × 10.

The comparative absence of these cells in adult life, as revealed by their scarcity in figure 3, indicates that they have migrated or died.

To speak of other cells of this type—glia cells—it is of particular interest to observe the numbers of them in the region where myelin is laid down at an early stage, i. e., between the olivary bodies. In over 10,000 cells in this locus from ten premature babies weighing between 1,500 and 2,500 Gm. (the average weight of the brain of nine of the patients being 250 Gm.) and in a similar number of cells in the same number of full term children weighing from 2,500 to 6,000 Gm. (the average weight of the brains of eight of the patients being 390 Gm.) it will be seen that there was a slightly higher percentage of oligodendroglia cells in the premature than in the mature infants (table).

Penfield [3] and others, notably Morrison,[4] have shown that this type of cell contains myelin in embryos. Apparently the period of usefulness for oligodendroglia cells ends in this respect after myelin is laid down, and perhaps the cells tend to disappear or become indifferent. Koch

*Comparison of Cells in Premature and Mature Infants*

| | Premature Infants, per Cent | Mature Infants, per Cent |
|---|---|---|
| Macroglia | 30 | 38 |
| Microglia | 15 | 14 |
| Oligodendroglia | 55 | 48 |

and Koch [5] viewed myelinization in the albino rat from the angle of growth and chemistry and found that it proceeded at a rapid rate from the tenth to the fortieth day after birth. Figure 4 shows the types of cells between the olivary bodies from a premature baby weighing 2,250 Gm., and figure 5 shows the amount of myelin laid down in that area in the same subject.

Another structure seen in these sections was that of portions of the choroid plexus of the fourth ventricle. So many controversies have been waged over what a normal choroid plexus is that we insert an illustration to show the structure on cross-section at this age. It has a peculiar delicacy. The epithelial covering of the inflected pia mater with its vascular supply may be of one or more layers. Figure 6 shows some of these tufts.

In figure 7 portions of three folia of the cerebellum are shown with cells at the free edge of the molecular layer. This appearance is always seen in the infant; sometimes it is visible until the age of 10 years

3. Penfield, W.: Brain **47**:430, 1924.

4. Morrison, R.: Tr. Am. Neurol. A., 1931, p. 444.

5. Koch, Waldemar, and Koch, M. L.: J. Biol. Chem. **15**:427. 1913.

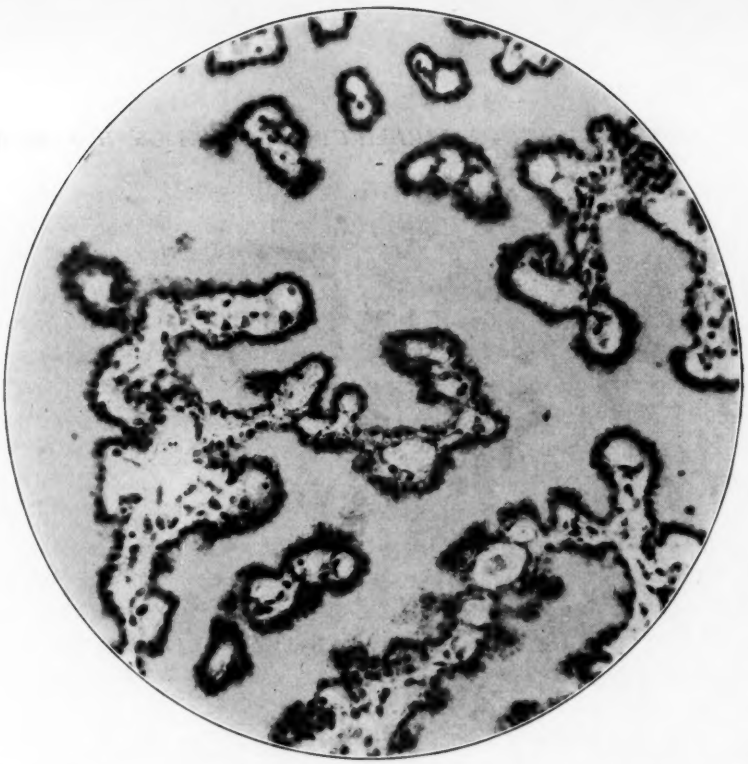

Fig. 6.—Section of a choroid plexus which was considered to be normal. Note the single layer of epithelial cells with excess layering at the focal points. Cresyl violet stain; × 215.

Fig. 7.—Section of folia of the cerebellum showing nonmigrating granule cells at the edge. Cresyl violet stain; × 70.

(Bailey), though not in every child over the age of 2 years (Gates). All of the eighteen sections containing cerebellum showed this non-migrating band of cells, as described by Bremer [6] and DeVries.[7]

## SUMMARY

The vessels seem inadequate in number to nourish as important a structure as the medulla oblongata.

In the new-born ependymal cells are of greater length than in older persons, averaging 20 microns.

Subependymal glia cells are more abundant in the new-born than in the aged.

Oligodendroglia cells are slightly more in evidence midway between the olivary bodies, where myelin is laid down at an early stage. In ten premature infants they represented 55 per cent of the cells; in ten babies born at term, 48 per cent.

Choroid plexuses may have various foci of heaping up of epithelial cells in their tufts.

Many cells are present at the surface of the folia of the cerebellum in the new-born.

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6. Stöhr, P., and Lewis, F. T.: Text Book of Histology, translated by J. L. Bremer, ed. 3, Philadelphia, P. Blakiston's Son & Co., 1930, p. 457.

7. DeVries, E.: Frühembryonale Ursache von Missbildungen des Nervensystems, Schweiz. Arch. f. Neurol. u. Psychiat. **19**:274, 1926.

# RESPIRATION IN INFANCY

## I. A METHOD OF STUDYING RATES, VOLUME AND CHARACTER OF RESPIRATION WITH PRELIMINARY REPORT OF RESULTS

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AND
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This study was undertaken for the purpose of obtaining some accurate information concerning the rate, character and volume of respiration in average normal infants in the first few months of life, and of learning what variations may normally be expected in the breathing of different infants and in that of the same infant at different times.

### METHOD AND APPARATUS

Since the use of a mask or nose-piece might be expected to disturb the infants and result in distorted types of breathing, respirations were recorded by an indirect method, similar to the method described by Murphy and Thorpe [1] and by Shaw and Hopkins.[2]

With this indirect method, the infant's body was enclosed in an air-tight chamber while the head remained outside the chamber, sealed off by a circular rubber collar, leaving the infant free to breathe the air of the room in a normal manner (fig. 1). Connected with the respiratory chamber was a spirometer which responded to displacements of air from the chamber by the expansion of the infant's body as he breathed. A writing point, attached to the spirometer float, traced the excursions of the spirometer on a kymograph and furnished a graphic record of the infant's respiration, the upstrokes representing inspiration and the downstrokes, expiration.

The respiratory chamber used in our investigation was a rigid cylinder, 2 feet (60.9 cm.) long and 14 inches (35.6 cm.) in diameter, made of 14 gage black sheet iron with welded seams and open at one end. We had previously attempted to use a square box made from thinner material, but found that one cylindric in shape and made from metal at least 14 gage thickness was necessary to insure complete rigidity.

Welded to the open end of the cylinder, making a frame about the opening, was a plate of 14 gage sheet iron, 16 inches (40.6 cm.) square, with a central

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From the Child Research Council and the University of Colorado School of Medicine.

1. Murphy, D. P., and Thorpe, E. S.: Breathing Measurements on Normal New-Born Infants, J. Clin. Investigation **10**:545 (Aug.) 1931.

2. Shaw, L. A., and Hopkins, F. R.: Respiration of Premature Infants, Am. J. Dis. Child. **42**:335 (Aug.) 1931.

circular aperture 14 inches in diameter. A detachable plate of the same material, with an aperture 9 inches (22.9 cm.) in diameter, was fastened against this frame by a rubber gasket and twelve wing nuts. For an infant in the first few months of life, it was not necessary to remove the detachable plate, as he could be inserted easily through the 9 inch circular opening.

The infant lay on his back, with the body resting on a padded board inside the chamber and the head outside the circular opening and resting on a trough-shaped head-rest.

The rubber collar used about the infant's neck for closure was the standard collar for the infant-size Drinker respirator. The collar was held in place between two pairs of crescent-shaped metal shutters clamped firmly against the front plate (fig. 1*B*). The inner pair of shutters was lined with a rubber gasket to insure an air-tight contact with the plate.

In addition to the opening at the front, three smaller openings led into the respiratory chamber. The first was a small stopcock valve at the rear end, which

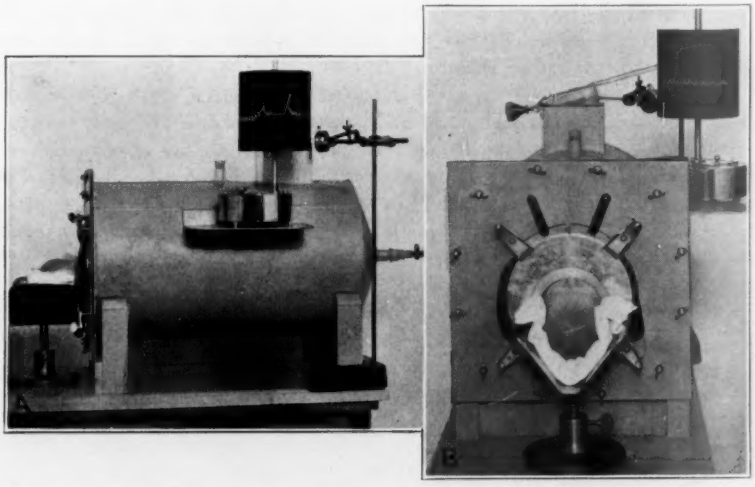

Fig. 1.—Respiratory machine with the infant in place. *A* is a lateral view; *B*, a front view showing the arrangement of the shutters.

was left open while the infant was inserted into the chamber, and which might be opened if it was necessary to make any adjustment of the level of the spirometer float while the infant was in place. At the top of the chamber was a second small opening through which a thermometer might be inserted for temperature determinations.[3] This opening was plugged with a rubber stopper when the thermometer was not being used.

The third opening was a rectangular aperture approximately 2 by 2¾ inches (5 by 7 cm.) which opened directly into the spirometer from the top of the respiratory chamber.

3. Temperature determinations for 10 consecutive infants showed that the average rise in the temperature of the respiratory chamber while the infant's body was enclosed in the chamber was less than 1 C.

Our spirometer, which was of the hinged-float type similar to the Krogh spirometer, was built directly on the upper surface of the respiratory chamber and formed an integral part of the chamber (fig. 1 *B*). We had previously tried using a separate spirometer which was joined to the chamber by means of a connecting tube of fairly small diameter. It was found, however, that the small outlet and the connecting tube led to undesirable pressure phenomena and to an accumulation of air within the spirometer.

The spirometer float was made of extremely thin nickel-silver and was exceedingly light and sensitive. The upper surface of the float was 10 cm. square. The submerged part had a circular curvature and was accurately counterbalanced by a screw weight at the center of curvature.

*Calibration of the Spirometer.*—According to the theoretical calibration, calculated from the dimensions of the spirometer and writing point, a vertical excursion of 1 mm. of the tip of the writing point represented a displaced volume of 2.31 cc. of air.

Not knowing whether our spirometer was sufficiently sensitive to make the actual equal to the theoretical performance, we undertook to check the calibration by measuring the excursions resulting from the displacement of a known volume of air from the respiratory chamber.

The first method which we tried consisted in displacing air from the chamber by introducing a supposedly known and constant volume of air from the outside. We attempted to deliver the known volume of air into the chamber by means of a syringe and also by a moving column of liquid in a buret, alternately introducing and withdrawing this air from the chamber in the hope of obtaining a tracing comparable to the records of the infants' respiration. We found, however, that the tracings obtained by these methods did not resemble those of infants' breathing; we were unable alternately to introduce and withdraw the air at a rate comparable even to the slowest respiratory rates observed in the infants, and the height of the excursions was so inconstant as to be useless for calibration.

After many unsuccessful attempts, our final solution of the problem of calibration was the construction by one of us (A. W.) of an "artificial baby" which displaced air from the respiratory chamber in a manner comparable to the displacement of air by our infant subjects.

This device (fig. 2 *A*) consisted of a 100 cc. B-D Luer syringe, mounted securely on a wooden base by means of the blocks *c*, *d* and *e*. The tip of the syringe from *a* to *b* represented the baby's head and neck, the rubber collar being attached at *b*, and the remainder of the apparatus rested inside the respiratory chamber. The head of the solid piston was embedded in a block (*g*), so that when a current of compressed air entered the syringe at *a* (outside the chamber) the piston moved back (inside the chamber) until the block *g* was stopped by the solid block *h*. Electric contacts at the point (*i*) set off a buzzer when the volume of displaced air was at the maximum. The Y-tube at *a* allowed interruption of the current of compressed air at the moment when the buzzer was set off. Elastic bands at *f* furnishead a simple and efficient means for the return of the piston to its original position. With this interrupted stream of compressed air the to-and-fro motions of the pistons might be as many as 30 a minute—a rate corresponding to the lower zone of the respiratory rates observed in the infants.

In order to produce not only a displacement of air inside the chamber but an up-and-down movement of the air comparable to that set up by the respiratory movements of a baby, a light wooden fan (*k*), mounted at the end of the device,

was worked synchronously with the piston by means of a jointed rod (*j*). Inside the chamber this fan was in approximately the same position as the baby's abdomen.

The "artificial baby" produced graphic records not unlike those of regular respiration (fig. 2*B*). The excursions in the tracings obtained in this way proved to be remarkably constant.

The volume actually displaced by the movement of the piston was calculated from careful measurements, with a vernier caliper, of the portion of the piston which actually moved in and out of the barrel of the syringe, and was found to be 36.75 cc.

By the technic used in the measurement of the respiratory volume, we measured the height of 100 excursions in records similar to that in figure 2*B* which were

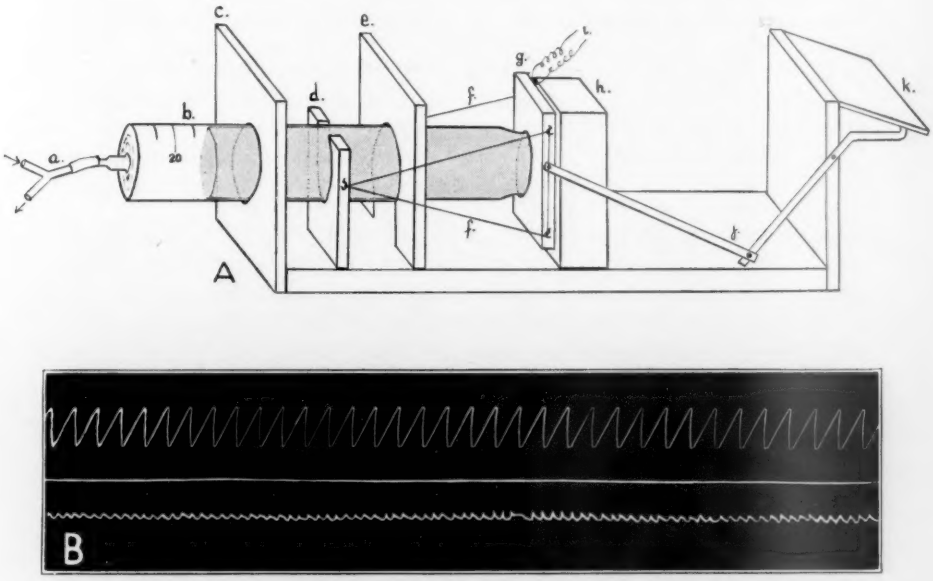


Fig. 2.—*A* is a diagrammatic drawing of the "artificial baby" used in the calibration of the spirometer. In the scheme, *a* indicates the Y tube for the alternate inlet and outlet of compressed air, and *b*, the attachment of the rubber collar, so that the portion *a-b* remains outside and the portion *b-k* inside the respiratory chamber; *c*, *d* and *e* are wooden blocks holding the barrel of the syringe firmly in place; *f* indicates the elastic bands which return the piston to the syringe when the compressed air is released, and *g*, the wooden block over the piston head; *h* represents the solid stop block; *i*, the buzzer contacts, which signal the maximum excursion of *g* between *e* and *h*; *j*, the jointed rod attached to *g*, and *k*, the light wooden fan moving synchronously with the piston. *B* is a record of the displacements of air by the moving piston inside the respiratory chamber of the apparatus shown in figure 2*A*.

obtained by repeated rapid displacements of the known volume of air. The height of the excursion in these records ranged from 15.8 to 16.1 mm., with an average height of 15.9 mm. The calibration factor was calculated by dividing the known

volume by the height of the excursion resulting from the displacement of this known volume, and for the values obtained in these determinations, was as follows:

$$\frac{36.75}{15.9} = 2.31.$$

This value was identical, to within two decimal places, with the theoretical calibration factor which was calculated from the dimensions of the spirometer.

It was concluded, therefore, that our spirometer was efficient to a degree which exceeded the limit of our accuracy of measurement, and that we were justified in using the theoretical calibration.

## EXPERIMENTAL MATERIAL AND TECHNIC

The subjects for this study were 27 average normal infants from the Salvation Army Home in Denver.

The ages of these infants, at the time when records of their respiration were obtained, varied from 20 hours to 13 weeks. Their weights ranged from 5½ pounds (2.5 Kg.) to 11½ pounds (5.3 Kg.). Nineteen of the infants were white; 3 colored, and 4, Mexican, and 1 was of combined Mexican and Filipino extraction. Seventeen of the group were boys and 10 were girls.

The infants were living under excellent environmental conditions in the nursery of the Salvation Army Home. Some were breast fed, and some, artificially fed with an evaporated milk formula. All appeared to be thriving.

Only 2 of the infants showed evidence of abnormality. In 1, a hematoma, the result of an injury at birth, was present in the right parietal region, and the infant was somewhat hypertonic and irritable. The other showed no external evidence of injury at birth, but was hypertonic and exceedingly irritable. Both these hypertonic infants were gaining normally, and looked healthy and well nourished in spite of their irritability. The records of the respiration of these 2 infants were essentially similar to those obtained from the other infants in the group.

All but 2 of the infants were supposedly born at full term. One of these was thought to have been born two weeks prematurely; the other was born about one month prematurely, but had done well from the first and seemed entirely normal except for size.

Sixty-nine records of respiration were obtained on different days from the 27 infants. From 5 of the infants, only 1 record was obtained; from 9, 2 records; from 8, 3 records; from 3, 4 records, and from 2, 5 records. The interval between obtaining the different records from the same infant varied from one day to three weeks. In most cases it was approximately one week. At least a portion of each of the 69 records consisted of tracings of respirations made while the infant was asleep. Some of the records also contained tracings made while the infant was awake and fairly quiet, and some, tracings of the respiration during crying.

All the records reported in this paper were obtained shortly after the infant's feeding at 10 a. m. A small portion of the feeding, for breast-fed infants a bottle of warm water, was always reserved to appease the infant if necessary after the rubber collar had been put on and the infant placed inside the respiratory chamber.

All clothing was removed from the infant in order to avoid any possible interference with the displacement of air from the chamber in response to the expansion of the infant's body.

For infants with fat necks the rubber collar sometimes proved to be uncomfortably tight. In these instances, we found that pulling the collar taut between

the two pairs of shutters relieved the pressure on the neck. When the collar was stretched in this way, it was important to make sure that it did not leave a leak under the infant's chin.

The behavior of the infants in response to our procedure was variable, the very young infants being, in general, easier to handle and pacify than the older infants of the group. We found that practically all the infants protested when the rubber collar was put on, and that some of them objected to being inserted into the chamber. In most instances, these grievances were soon forgotten when the infant was given a small amount of the evaporated milk or of water. If an atmosphere of undisturbed quiet was maintained in the laboratory and we were willing to wait patiently, the infants almost always fell asleep in the respiratory chamber.

The infant was left in the chamber until a record consisting of several satisfactory tracings of the respiration had been obtained. In most cases the time did not exceed one-half hour.

## ANALYSIS OF RECORDS

The outstanding characteristic of our 69 records of the respiration in normal young infants was the extreme variability of type, rate and volume—not only in the breathing of different infants but in that of the same infant on different days and often even from minute to minute during continuous sleep.

*Character of Respiration During Sleep.*—Some degree of variation during sleep was observed in the breathing of nearly all the infants, many of the records showing two or more distinct types in different portions of the record, as well as decided fluctuations in rate and volume. An extreme example was seen in a record obtained from a full term, normal infant on the fourth day of life. During a brief period of continuous sleep, we obtained from this infant excellent tracings of each of the three principal types of breathing which we shall describe and also of a bizarre type unlike any of the more usual forms. The respiratory rate during this period fluctuated between 27 and 41 per minute. The average depth of the inspiration varied from 11 to 21 cc. with the different types of breathing. The minute volume fluctuated between 354 and 569 cc.

With 2 exceptions, the respiration during sleep as shown in our tracings may be classified into three main types. The first was a regular type, similar to normal adult respiration, but with a more rapid rate (fig. 3 *A*). In this type the duration of inspiration and that of expiration were approximately the same; no pronounced pause took place between breaths; the rate was fairly uniform, and most of the inspiratory excursions were of approximately equal amplitude, though single excursions were occasionally considerably smaller or larger than the average excursion.

The second was a slow type, in which a quick inspiration was followed by a greatly prolonged expiration of interrupted or cog-wheel character (fig. 3 *B*). There was no appreciable pause between the end of one breath and the beginning of the next. The rate, as in the first type, was approximately uniform. However, on account of the prolonged expirations, the rates observed in this type of breathing were considerably slower than those usually observed in the regular type. The amplitude of the inspiratory excursions in this type was sometimes fairly uniform, and sometimes showed a good deal of variation.

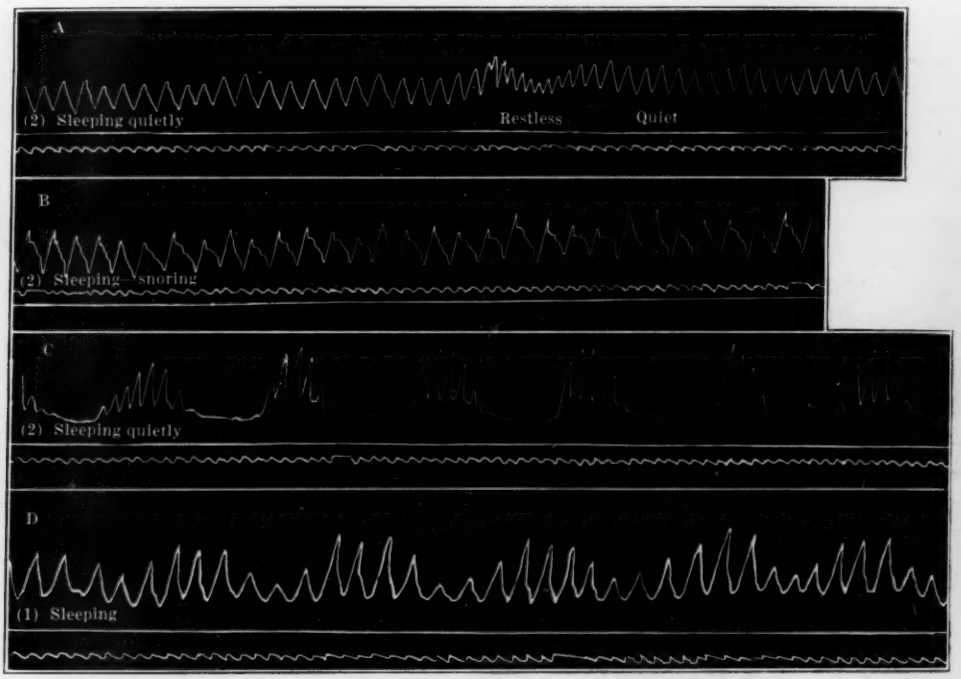


Fig. 3.—Tracings of the principal types of breathing observed during sleep. *A* is a record of the regular type of respiration with fairly rapid rate, obtained from a boy 12 days old, weighing 8¼ pounds (3,742.5 Gm.); *B*, a record of the slow type with prolonged cog-wheel expiration, obtained from a boy 5 weeks old, weighing 8 pounds (3,628 Gm.); *C*, a record of the extremely periodic type, resembling Cheyne-Stokes' respiration, obtained from a boy 24 days old, weighing 8½ pounds (3,855.5 Gm.), and *D*, a record of the moderately periodic type, obtained from a girl 4 weeks old, weighing 9 pounds (4,082.3 Gm.).

The third type of breathing observed from our records was the periodic, in which the amplitude of the inspiratory excursions waxed and waned in recurring cycles. This type may be divided into two sub-

types. The first was an extremely periodic type, resembling Cheyne-Stokes' respiration, in which the periods were separated by intervals of complete apnea, often of several seconds' duration (fig. 3 *C*). The second subtype was a moderately periodic type, in which cyclic variations in amplitude recurred without an interval of apnea (fig. 3 *D*).

Minor individual variations were observed within each of these principal types of respiration. In 2 instances, tracings were obtained of a combined type, in which the rhythm was moderately periodic while the individual respirations were of the slow type with prolonged cog-wheel expiration. The 2 tracings which we were unable to classify showed bizarre forms of breathing, which were completely irregular, with extreme variations in the character of the individual respirations, the rapidity of the breathing and the amplitude of the inspiratory excursions (fig. 4). The breathing was interrupted at irregular intervals by a prolonged pause, which occurred sometimes at the end of inspiration but more often at the end, and occasionally in the middle of, expiration. Both

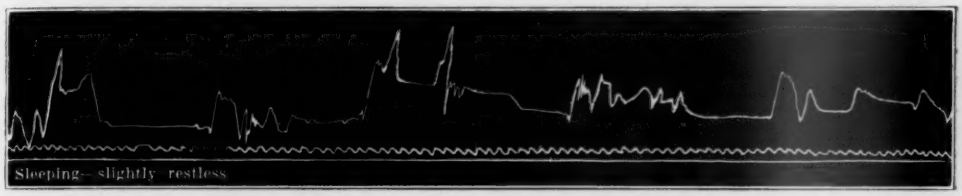


Fig. 4.—Tracing of a bizarre form of breathing observed in a full term normal infant, aged 4 days.

samples of this bizarre type of irregular respiration were obtained from infants in the first few days of life and during slightly restless sleep.

In the distribution of the various types of respiration among the 69 records, considerable overlapping was found, owing to the fact that many of the records contained samples of more than one type. The regular type was observed in 43 records, the periodic type in 38, the slow type with cog-wheel expiration in 12 and the unclassified bizarre forms in 2.

When the records were distributed according to the ages of the infants expressed in weeks, it was found that the regular and the periodic type were common at all ages from birth to 13 weeks, which was the age of the oldest infant in our group, and that in each age group observed these two types occurred with almost equal frequency. The slow type with prolonged expiration was less common and appeared to be confined to the early weeks of life. Its highest incidence in our records was noted in the samples obtained from infants in the first week of life. We did not observe this type in any of the infants past 6 weeks of age. The

unclassified, completely irregular types of breathing were observed only in the first week of life.

No particular correlation was observed between the type of breathing and the body weight.

Our records showed a slight difference between the two sexes in regard to the distribution of the types of breathing, the relative frequency of the regular type being somewhat greater among the boys, and that of the periodic type somewhat greater among the girls. In the 49 records obtained from boys, the regular type was observed in 33 (67 per cent) and the periodic in 26 (53 per cent). In the 20 records obtained from girls, the regular type was observed in 10 (50 per cent) and the periodic in 12 (60 per cent).

No correlation was observed between the disposition of the infant and the type of breathing. We found, however, that when an infant was disturbed during sleep, or when his sleep was interrupted by an interval of fussing, the type of respiration was often different following the disturbance. In some instances the change was from the regular to the periodic type. In others breathing which had been periodic became regular.

The respiration of the 2 premature infants in our group did not differ noticeably from the respiration of the full term infants so far as type of breathing was concerned. In the case of one of these, a white girl born about one month prematurely, who was in the eighth week of life at the time of the first record, the regular type appeared in 2 records and the periodic in 1. The other, a colored boy born about two weeks prematurely, who was 4 days old at the time of the first record, showed exclusively the regular type of breathing. Incidentally, the 3 colored infants in our series showed only regular breathing.

*Comparison with Types of Respiration During Sleep Described in the Literature.*—Descriptions in the literature of respiration in infancy include those of all the types observed in our records; not all these types, however, have been reported as occurring in normal, healthy infants.

The periodic type was observed occasionally by Murphy and Thorpe [1] in normal new-born infants, usually as a transitory phenomenon at the beginning of sleep. Peiper [4] reported the observation of periodic types of breathing in premature infants and occasionally in full term, new-born infants. He did not consider this type necessarily indicative of disease, though he observed it frequently in certain pathologic con-

4. Peiper, A.: Die Atemstörungen der Frühgeburten, Ergebn. d. inn. Med. u. Kinderh. **40**:1, 1931.

ditions. Vormittag [5] noted periodic breathing in a number of healthy infants. In many instances, it was only transitory, but in 2 infants whose histories were followed to the age of 2½ months, he observed this type exclusively. Our own findings seemed to indicate that the periodic type of respiration may be much more frequent and less transitory in normal young infants than has been previously described.

The slow type of breathing with prolonged cog-wheel expiration (fig. 3*A*) which we observed corresponds to the type described as *Keuchatmung* by Peiper.[4] Peiper noted this type in certain moribund premature infants and considered its presence always a bad sign. He described a groaning sound accompanying each expiration. Vormittag observed *Keuchatmung* in 18 healthy infants. He did not note the groaning expiratory sound described by Peiper. In our own group of infants this type of breathing occurred rather frequently in the early weeks of life and seemed in no way indicative of an abnormal condition. In 2 instances we observed a slight snoring sound accompanying the prolonged expirations.

The unclassified bizarre forms of breathing observed by us in 2 new-born infants (fig. 4) are similar to the completely irregular breathing, with long pauses, which Peiper noted in a few premature infants, all of whom were moribund. The 2 infants in our group who showed this bizarre breathing were observed subsequently for about one month and seemed at all times to be normal and healthy.

There was not observed in any of our infants the gasping type of breathing which Peiper and Vormittag described as *Schnappatmung*.

The type described by Vormittag as a mirror image of *Keuchatmung*—the inspiration instead of the expiration being prolonged and interrupted—was not observed as a sustained type. We did, however, occasionally note single inspirations of this character, to be seen chiefly in records obtained during restless sleep or while the infant was awake.

*Rate of Respiration During Sleep.*—There were selected for detailed study of the rate and volume 118 minute samples of respiration during sleep from representative portions of the 69 records. The average rate for the whole group was 41 per minute. The range in the rate of respiration was from 24 to 76 per minute, with the following distribution:

| Rate of Respiration | Samples |
|---|---|
| 20 to 29 | 21 |
| 30 to 39 | 27 |
| 40 to 49 | 49 |
| 50 to 59 | 18 |
| 60 to 69 | 2 |
| More than 70 | 1 |

5. Vormittag, St.: Untersuchung über die Atmung des Kindes, Monatschr. f. Kinderh. **58**:249 (July) 1933.

When the samples were distributed according to the types of breathing, there was a distinct correlation between the respiratory rate and the type, as shown in the following tabulation:

| Type of Breathing | Range of Rates | Average Rate |
|---|---|---|
| Regular | 25 to 56 | 45 |
| Slow, with cog-wheel expiration | 24 to 31 | 27 |
| Periodic (including both types) | 24 to 76 | 40 |
| Extremely periodic | 24 to 48 | 33 |
| Moderately periodic | 30 to 76 | 43 |
| All types combined | 24 to 76 | 41 |

The average rate computed for the samples of regular respiration was higher than that for the whole group of samples, though there was a considerable range of variation in the rates observed in this type of breathing. The consistently low rates observed in the samples showing cog-wheel expiration resulted from the time consumed by the prolonged expirations in this type. The periodic type, when both subtypes were included, showed the greatest range of variation in rate, with an average rate essentially the same as that for the whole group of samples. When the samples of periodic breathing were distributed between the extremely and the moderately periodic forms, we noted a striking difference in the average rates for the two subtypes. The low rates observed in samples of the extremely periodic type were the result of the prolonged intervals of apnea between the periods.

When the 118 samples were distributed according to the ages of the infants, from birth to 13 weeks, no correlation was found between the respiratory rate and age, the average rate being nearly the same in each week of life. Likewise no correlation was shown in our samples between the respiratory rate and the body weight.

A slight difference was observed in the respiratory rate according to sex, the incidence of slow rates appearing relatively greater in the 31 samples obtained from girls and the incidence of rapid rates, greater in the 87 samples obtained from boys. The 12 most rapid rates appeared in samples obtained from boys. The average rates for each sex group are shown in the following tabulation:

| Sex | Range of Rates | Average Rate |
|---|---|---|
| Male | 25 to 76 | 43 |
| Female | 24 to 51 | 36 |
| Both sexes | 24 to 76 | 41 |

No correlation was observed between the respiratory rate and the disposition of the infant. The rate, however, tended to be more rapid when the infants were sleeping lightly or were somewhat restless, and to become slower as sleep became more profound.

The 7 samples obtained from our 2 premature infants showed respiratory rates consistently higher than the average rate for the group. The rates observed in these samples ranged from 43 to 64 per minute, the average rate being 50 per minute.

A comparison of the respiratory rates observed in our group of infants with some of the rates reported in the literature is shown in table 1.

*Volume Inspired During Sleep.*—Determinations of volume were made from each of our 118 minute samples by measuring the vertical

TABLE 1.—*Rate of Respiration in Healthy Young Infants During Sleep as Reported in the Literature*

| Author | Description and Age of Infants | Range of Rates | Average Rate |
|---|---|---|---|
| Murphy and Thorpe | First week | 24 to 116 | 43 |
| Pelper | Healthy, premature | Not stated | Between 35 and 45 |
| Shaw and Hopkins | Healthy, premature | 31 to 114 | 58 |
| Vogt: Monatschr. f. Kinderh. **42**: 460, 1929 | First week | 27 to 48 | 36 |
| Vormittag | First week | 22 to 72 | 35 |
| Vormittag | Second to fourth week | 21 to 71.5 | 37.7 |
| Vormittag | One to six months | 21 to 58 | 33.3 |
| Our infants | First to thirteenth week | 24 to 76 | 41 |

TABLE 2.—*Results of Volume Determinations Based on 118 Minute Samples*

| | | |
|---|---|---|
| **Minute Volume of Inspired Air** | | |
| Range | | 354.4 to 2,607.4 cc. |
| Average | | 1,078.3 cc. |
| Distribution: | | |
| Minute Volume, Cc. | | Samples |
| Less than 500 | | 3 |
| 500 to 1,000 | | 43 |
| 1,000 to 1,500 | | 62 |
| 1,500 to 2,000 | | 9 |
| More than 2,000 | | 1 |
| **Minute Volume per Pound (453.59 Gm.) of Body Weight** | | |
| Range | | 59.1 to 230.7 cc. |
| Average | | 131.3 cc. |
| Distribution: | | |
| Minute Volume, Cc. | | Samples |
| 50 to 100 | | 15 |
| 100 to 150 | | 77 |
| 150 to 200 | | 21 |
| 200 to 250 | | 5 |
| **Average Depth of Inspiration** | | |
| Range | | 9.7 to 53.1 cc. |
| Average for the group | | 27.3 cc. |
| Distribution: | | |
| Minute Volume, Cc. | | Samples |
| Less than 10 | | 1 |
| 10 to 19 | | 18 |
| 20 to 29 | | 60 |
| 30 to 39 | | 32 |
| 40 to 49 | | 5 |
| More than 50 | | 2 |

height of each inspiratory excursion in the sample. The measurements were made with a vernier caliper graduated in intervals of 0.1 mm. The distance was measured from a horizontal base-line to the low point before the beginning of the excursion and to the high point at the peak. The difference between these measurements gave the vertical height of the excursion. The sum of the vertical heights of all the excursions

in the sample, expressed in millimeters, multiplied by our calibration factor (2.31), gave the minute volume in cubic centimeters. The average height of excursion, found by dividing the sum of the vertical heights by the number of excursions in the sample, was multiplied by the calibration factor (2.31) to give the average depth of inspiration. All measurements and calculations were made by the same person (J. D.).

The results of the volume determinations based on our 118 minute samples of respiration during sleep are shown in table 2.

When the 118 minute samples were distributed according to the types of breathing, less correlation was found between the minute volume and the type than between the respiratory rate and the type. With the exception of the slow type with prolonged expiration, the various types of breathing differed but little in their average minute volume. The small minute volumes observed in the slow type were dependent partly on the slow rates consistently observed in this type of breathing and partly on the occurrence of this type for the most part in the youngest infants of the group.

The average minute volumes for each type are shown in the following tabulation:

| Type of Breathing | Average Minute Volume, Cc. |
|---|---|
| Regular | 1,097.0 |
| Slow, with "cog-wheel" expiration | 775.3 |
| Periodic (including both subtypes) | 1,138.9 |
| Extremely periodic | 1,116.2 |
| Moderately periodic | 1,156.4 |
| All types combined | 1,078.3 |

The fact that the average minute volume was relatively constant, as compared with the respiratory type and rate, may be explained in part by the roughly inverse relation frequently observed between the average depth of respiration and the respiratory rate. This is well illustrated by a comparison of the samples of tracings of the two kinds of periodic breathing as to rate, average depth and minute volume. As is shown in the following tabulation, the average rate and depth in the two types were inversely proportional and the differences in rate and depth were relatively much greater than those in the minute volume.

| Type | Average Rate | Average Depth of Inspiration, Cc. | Average Minute Volume, Cc. |
|---|---|---|---|
| Extremely periodic | 33 | 34.6 | 1,116.2 |
| Moderately periodic | 43 | 26.0 | 1,156.4 |
| Ratio of moderately to extremely periodic type | 1.33 : 1 | 1 : 1.33 | 1.06 : 1 |

When the 118 samples were distributed according to the ages of the infants, a tendency was observed for the minute volume to increase with increasing age, though the correlation was by no means absolute.

The following tabulation shows the average minute volume for each week of life for which we had samples of respiratory tracings:

| Week of Life | Average Minute Volume, Cc. |
|---|---|
| 1 | 626.3 |
| 2 | 879.4 |
| 3 | 923.7 |
| 4 | 896.4 |
| 5 | 1,098.0 |
| 6 | 1,101.9 |
| 7 | 1,231.3 |
| 8 | 1,335.9 |
| 9 | 1,154.2 |
| 10 | 1,228.0 |
| 13 | 1,661.4 |

The increase in the minute volume with that of age was proportionately much more rapid than the increase in body weight with that of age. This resulted in a sharp rise, as age increased, in the value of the minute volume per pound of body weight. The average minute volume per pound (453.59 Gm.) as observed in our records increased from 94.9 cc. in the first week of life to 174.5 cc. in the thirteenth week.

No correlation was observed between the minute volume and the sex, the average minute volume for each of the two sexes being almost identical with that of the other and with the average minute volume for the whole group. The average depth of inspiration was greater in the samples obtained from girls than in those from boys. This again illustrates well an inverse relation between the depth of inspiration and the respiratory rate, with the minute volume remaining constant.

| Sex | Average Rate | Average Depth of Inspiration, Cc. | Average Minute Volume, Cc. |
|---|---|---|---|
| Male | 43 | 25.9 | 1,078.5 |
| Female | 36 | 31.0 | 1,077.5 |
| Both sexes | 41 | 27.3 | 1,078.3 |

No correlation was observed between the minute volume and the disposition of the infant or between the minute volume and the racial extraction.

The 7 samples obtained from our 2 premature infants showed minute volumes which differed but little from the values for the full term infants of the same age, in spite of the smaller size of the premature infants. The average minute volume per pound for the premature infants is 153.6 cc., as compared with the value of 131.3 cc. for the whole group.

*Comparison with the Minute Volumes Reported in the Literature.*—Respiratory volumes, obtained by a method similar to ours, were reported by Murphy and Thorpe [1] and by Shaw and Hopkins.[2] Murphy and Thorpe, working with 50 normal infants in the first few days of life, obtained the following values for respiration during sleep:

| | |
|---|---|
| Minute Volume, Cc. | |
| Range | 433 to 1,413 |
| Average | 721.4 |
| Average Depth of Inspiration, Cc. | |
| Range | 10 to 27 |
| Average | 16.7 |

Our own determinations for infants during the first week of life gave somewhat similar results, with an average minute volume

for this age group of 626.3 cc. and average depth of inspiration of 17.1 cc.

Shaw and Hopkins obtained 20 records of respiration from 9 premature infants who were in good health and reported the following observations:

| | |
|---|---|
| **Minute Volume, Cc.** | |
| Range | 304 to 1,225 |
| Average | 698 |
| **Minute Volume per Pound of Body Weight, Cc.** | |
| Range | 121.0 to 273.8 |
| Average | 193.1 |

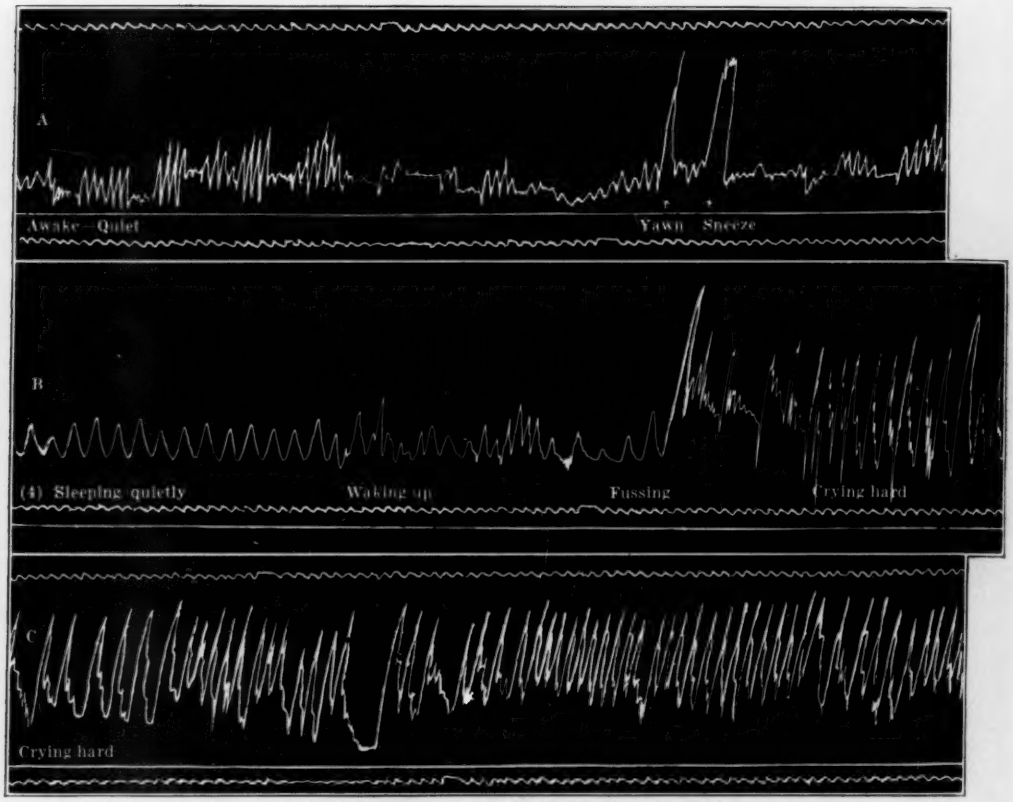


Fig. 5.—Tracings of breathing in infants while awake. *A* records respirations during quiet breathing, yawning and sneezing in a boy approximately 20 hours old, weighing 6¾ pounds (3,061 Gm.); *B*, respirations while waking from sleep, fussing and crying, in a girl 32 days old, weighing 8½ pounds (3,855.5 Gm.), and *C*, respirations during hard crying.

In the 2 premature infants in our own series, the minute volumes ranged from 914.8 to 1,328.5 cc., with an average of 1,034.4 cc. The minute volume per pound for our premature infants ranged from 92.2 to 212.6 cc., with an average of 153.6 cc.

*Respiration While Awake and Quiet.*—Twenty-six samples were obtained for minute and one-half minute periods from infants who were awake and fairly quiet. These samples showed an even greater variability than did those obtained during sleep (fig. 5 *A* and *B*). The breathing during the waking state could not be classified into the clearcut types observed in sleeping infants, though many of the samples showed tracings suggestive of one or another, or of combinations, of the types of breathing observed during sleep. The respiratory rates observed in the waking infants were considerably more rapid than those for the sleeping infants, ranging from 46 to 108 per minute, with an average rate of 59 per minute. The minute volume, likewise, was usually much greater in the waking infants, ranging from 555.8 to 3,213.7 cc., with an average of 1,319.5 cc.

*Respiration During Crying.*—Sixteen samples (for minute and one-half minute periods) were obtained from infants while crying. The rates ranged from 40 to 114 per minute, with an average of 68 per minute.

Determinations of volume from samples obtained during periods of crying were necessarily somewhat less accurate than those obtained during sleep, because the kicking and struggling of the crying infant jarred the spirometer. It is obvious, however, that respiration during crying is characterized by a striking increase in the average depth of inspiration and in the minute volume of air inspired (fig. 5 *B* and *C*). In our 16 samples the minute volume during crying ranged from 1,551.4 to 5,943.9 cc., with an average of 3,535.3 cc.

## SUMMARY

A method of studying the rate, volume and character of respiration in young infants is described.

An analysis of 69 records of respiration obtained by this method from a group of average normal infants yielded the following information:

1. The respiration of average normal infants in the first three months of life is characterized by an extreme variability in the types of breathing as well as in the respiratory rate and in the volume of air inspired.

2. The respiration shown in records obtained during sleep may be classified into three main types.

3. The respiratory rate shown in records obtained during sleep ranged from 24 to 76 per minute, with an average of 41 per minute.

4. The minute volume of air inspired during sleep ranged from 354.4 to 2,007.4 cc., with an average of 1,078.3 cc.

5. The minute volume per pound of body weight during sleep ranged from 59.1 to 230.7 cc., with an average of 131.3 cc.

6. The average depth of inspiration during sleep ranged from 9.7 to 53.1 cc., with an average of 27.3 cc.

7. Certain differences in respiration according to age and sex are suggested by the records obtained during sleep.

8. The minute volume tends to be relatively more constant than either the rate or the depth of respiration.

9. The respiratory rate and the average depth of inspiration often vary inversely.

10. The respiration of waking infants is more variable and more rapid than that during sleep and is characterized by a greater minute volume of inspiration.

11. The respiration during crying is characterized by a marked increase in the depth of inspiration, in the minute volume of inspired air and usually in the rate as well.

It is suggested that the soundness of this method and the building up of normal standards may pave the way for the acquisition of much valuable data concerning the changes in respiration in certain diseases of infancy.

# INCIDENCE OF SPINA BIFIDA OCCULTA IN CHILDREN WITH AND WITHOUT ENURESIS

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Enuresis nocturna is usually considered to be present when a child after reaching the age of 3 years continues bed wetting. The age limit is an arbitrary one. There are many children as young as 1½ or 2 years of age who without any special training acquire control of the bladder and sleep through the night without bed wetting. Other children may require to be awakened once during the night to empty the bladder. Some children require a period of training in order to gain sphincteric control. Other children in spite of the most rigorous measures and apparent cooperation on the part of the parents and the child continue to be bed wetters. The problem is usually considered a functional one, a behavior disorder coming within the province of a child guidance clinic, and requiring instructions for the mother and the placing of the child on his own responsibility. The period of training is frequently prolonged, and in many instances, in spite of apparently good cooperation on the part of the parents as well as the child, the treatment results in failure.

In this study an attempt is made to explain certain cases of enuresis in children on an organic basis. Every child coming to the child guidance clinic of the fifth division of the Neurological Institute and the neurological department of the Vanderbilt Clinic receives a complete neurologic examination. For the last year every child with enuresis, in addition to being given the general neurologic examination was referred as a routine for roentgen examination of the spine of the lumbosacral region in order to determine the presence or absence of spina bifida occulta.

Spina bifida is a developmental defect of the spinal column, which presents a failure of fusion between the various constituent parts of the vertebrae with or without protrusion of the spinal cord or its mem-

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The roentgenographic studies and reports were made by Dr. Paul C. Swenson of the Presbyterian Hospital and Dr. John Caffey of the Babies' Hospital.

branes. There are various classifications of spina bifida. For practical purposes the following types may be considered:

1. Complete rachischisis, in which the defect extends the entire length of the vertebral column.

2. Partial spina bifida, in which the defect involves one or more vertebrae. While in the majority of cases the failure of fusion involves the dorsal portion of the vertebrae, i. e., the neural arches, and is known as dorsal spina bifida, it may, although very rarely, occur ventrally, involving the vertebral bodies and being known as ventral spina bifida. The defect usually occurs in the lumbosacral region, but it may occur in the cervical portion of the vertebral column.

Under partial spina bifida three subdivisions may be considered.

(*a*) Myelomeningocele, in which the spinal cord and its meninges protrude through the bony defect.

(*b*) Meningocele, in which only the meninges protrude through the bony defect.

(*c*) Spina bifida occulta, in which there is a bony defect without any protrusion of the spinal cord or meninges.

Previous to the introduction of the x-ray, spina bifida occulta was diagnosed by the presence of certain conspicuous changes over the skin in the region of the defect. The most frequent alteration is a hypertrichosis in which the hair shows a tendency to a concentric distribution. There may be simply a dimple or an indentation called "fovea coccygea," or there may be certain cicatricial changes, pigmented spots or a telangiectasis. Since the introduction of the x-ray, however, it has been demonstrated that many patients showing the aforementioned changes in the cutaneous surface may be entirely without spina bifida occulta, and the diagnosis is now made conclusively on the evidence disclosed by the x-ray picture of nonfusion in the bodies or laminae of the vertebrae, which present the appearance of linear or transverse fissures or gaps of variable extent in these bony structures.

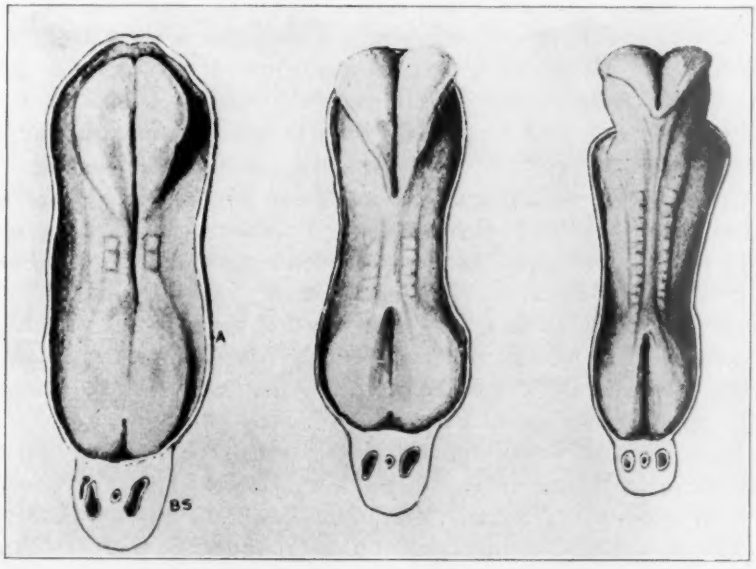
For a clearer understanding of the problem, it is important to refer briefly to the embryology of the structures involved and to the origin of spina bifida occulta.

In general, while the spinal cord is ectodermal in origin, the various coverings of the cord and the vertebral column are mesodermal in origin.

Along the dorsum of the embryonic disk there appears an elongated plate of thickened ectoderm known as the neural plate. The ectodermal band becomes thickened, a groove appears along the midline, and the margins are raised above the surface of the embryo, forming the neural groove. The margins, known as the neural or medullary folds, continue to push upward and finally meet and fuse with each other throughout the entire length in the middorsal line, forming the neural tube.

The fusion of the medullary folds to produce the neural tube begins in the middle region of the plate and then proceeds both cephalad and caudad. According to His, this process is completed in the second or third week of embryonic development, when the embryo is from 2 to 3 mm. in length. The fact that the closure of the neural tube is delayed longest in the caudal and cephalic ends may account for the fact that spina bifida occurs mainly in the cervical and lumbosacral regions; spina bifida of the thoracic region is rare.

Ultimately, the neural tube breaks away from the overlying surface ectoderm. Almost parallel with the process described in the preceding




Three views of the embryonic area at different and successive times, to show the elongation of the groove, its closure with formation of neurospores and the development of somites. *BS* indicates the body stalk; *A*, the cut edge of the amnion. After Fraser, J. E.: A Manual of Embryology, New York, William Wood & Company, 1932, p. 47, fig. 24.

paragraph, the mesoblast intrudes itself between the neural tube and the overlying skin, forming the primitive segments or somites which gradually become transformed into the future vertebral column that surrounds the medullary tube.

It is evident that any disturbance which prevents the closure of the neural tube will at the same time interfere with the normal development of the vertebrae around that part of the cord. This indicates that the cause of spina bifida is ectodermal in origin.

The question whether spina bifida is ectodermal or mesodermal in origin has interested many investigators. Von Recklinghausen [1] was of the opinion that, as a result of a fault in mesodermal development, the medullary tube did not detach itself from the superficial ectoderm and its proper closure was therefore prevented, the primary cause for the defect being mesodermal in origin. Ernst [2] expressed the belief that the primary maldevelopment is ectodermal in origin, and that the bony defects are secondary to a developmental defect in the spinal cord. Keiller,[3] in the classification of spina bifida from an embryologic point of view, placed spina bifida occulta in the group in which the gross defect is limited to mesoblastic structures, but immediately supplemented this by a statement that probably in the more severe cases the causative factor is primarily ectodermal in origin. According to this classification two forms of spina bifida occulta are distinguished. The simplest form is one in which the spines of one or more vertebrae are bifid. The more serious anomaly is that in which the laminae (neural arches) are incompletely formed. Winkler,[4] from a clinical point of view, taking into consideration the variable symptomatology of spina bifida occulta, concluded that the defect is due to a faulty development of both the ectoderm and the mesoderm. It is evident that most investigators have agreed that the defect of development that produces spina bifida is primarily some defect in closure of the neural tube. While this defect in closure of the neural tube may be only transitory, with no lasting anomaly in the fully developed spinal cord, it leaves its mark in the defect of the vertebrae in that area.

Since spina bifida occulta is diagnosed roentgenographically on the presence of a fissure or gap in the bony structure of the vertebrae (usually in the neural arches), the question naturally arises whether the defect may not be due simply to a failure of ossification. It is difficult to understand why the neural arches which have developed normally through the membranous and cartilaginous stages should suddenly fail to ossify, and why this failure of ossification should occur mainly in the lumbosacral or cervical region, regions which embryologically are the last to close. Minot [5] stated that ossification is a supplementary

1. von Recklinghausen, F.: Untersuchungen über die Spina bifida, Virchows Arch. f. path. Anat. **105**:243, 1886.

2. Ernst, P.: Missbildungen des Nervensystems, in Schwalbe, Ernst: Die Morphologie der Missbildungen der Menschen und der Tiere, Jena, Gustav Fischer, 1909, vol. 3, p. 72.

3. Keiller, V. H.: A Contribution to the Anatomy of Spina Bifida, Brain **45**:31, 1922.

4. Winkler, W.: Die Klinik der Spina bifida occulta, Deutsches Arch. f. klin. Med. **166**:303, 1930.

5. Minot, C. S.: Human Embryology, New York, William Wood & Company, 1892.

process and produces no important change in the form of anatomic relations of the vertebrae. Joseph [6] stated that the causative factors in congenital defects have their inception during the evolutionary period of the membranocartilaginous stage. Seldom can faulty ossification be held responsible, for this process has no choice except to follow the path laid out for it by the membranocartilaginous stages.

As to the actual causes that produce this defect of development in the medullary tube there are various theories. The earlier view (von Rokitansky, Virchow) was that this developmental defect was due to an accumulation of spinal fluid within the central canal which prevented the union of the edges of the neural plate to form the neural tube and at the same time interfered with the proper development of the structures which immediately surround the neuraxis. The main support for this theory is that spina bifida is often associated with hydrocephalus. The principal objection to the theory is based on the fact that the choroid plexus does not begin to secrete fluid until about the tenth week, whereas all writers agree that the inception of the process which leads to the production of spina bifida must take place no later than the third week. Frazier [7] believed that arrested development is responsible for one phase of the anomaly, increased cerebrospinal pressure for the other, the increased pressure due either to abnormal secretion or to delayed absorption of the cerebrospinal fluid. Lebedeff [8] believed the defect to be due to the bending backward of the embryo causing an abnormal stress on the dorsal wall and preventing the closing of the medullary tube and its coverings. Experimentally it has been possible to produce spina bifida at will in some of the lower vertebrates by treating the developing egg with a solution of sodium chloride (Hertwig). At the same time other defects in the nervous system (anencephaly) were produced. In these experiments the malformations followed a retarded closure or lack of closure of the neural tube.

The symptoms of spina bifida occulta depend on the extent of the involvement of the spinal cord and nerve roots. The symptoms may be present at birth or they may come on at puberty or later, often following some accident, fall or injury. This is important to bear in mind, because occasionally a child may suddenly present enuresis at the age of 9 or 10 years although until that time there was perfect control of the bladder. Spiller [9] stated that the injury may be the result only of an excessive

6. Joseph, B.: A Study of Mesorachischisis (Spina Bifida Occulta), Am. J. Dis. Child. **5**:285 (April) 1913.

7. Frazier, C. H.: Surgery of the Spine and Spinal Cord, New York, D. Appleton & Company, 1918.

8. Lebedeff, A.: Ueber die Entstehung der Anencephalie und Spina bifida bei Vögeln und Menschen, Virchows Arch. f. path. Anat. **86**:263, 1881.

9. Spiller, W. G.: Enuresis, Am. J. M. Sc. **151**:469, 1916.

bending of the trunk forward on the legs causing stretching of the lower sacral roots or it may follow such exercise as bicycle riding. Altschul [10] stated that external influences which under ordinary circumstances would not result in the development of symptoms may produce enuresis in persons who possess an inherent defect in the vertebral column. The most important feature is weakness of the sphincters, which may involve both the rectum and the bladder or the latter alone. This may be accompanied by disturbances of sensation, especially the appreciation of differences in temperature particularly in the feet and more frequently in the toes; abnormal cutaneous and tendon reflexes, which may be either subnormal or absent, and congenital defects in the feet, such as clubfoot, trophic disturbances of the toes and syndactylism between the second and third toes, usually bilateral. Other anomalies may be present, such as the hypertrichosis of the sacral region which has been mentioned, lipoma in the coccygeal region or simply a depression over the sacrum. Roentgen examination of the spine will reveal a defect in the vertebrae in the lumbosacral region.

Virchow,[11] in 1875, was the first one to employ the term "spina bifida occulta." He called attention to its association with lumbosacral hypertrichosis. Knowledge of the possible relationship of enuresis and spina bifida occulta dates from 1909, when Fuchs [12] made systematic investigations of recruits in the Vienna garrison who suffered from enuresis. Twenty-four recruits were examined, and as a result of these examinations he described a clinical syndrome named by him "myelodysplasia." Riley,[13] quoting Fuchs, stated that this syndrome was characterized by (1) sphinteric weakness, (2) syndactylism of the second and third toes, (3) syringomyelic sensory disturbances, (4) spina bifida, (5) abnormal skin and tendon reflexes of the abdomen and lower extremities, (6) deformities of the feet, with trophic and vasomotor disturbances, (7) skin changes, hypertrichosis, nevus and fovea coccygea. Mattauschek,[14] who examined the recruits with Fuchs, stated that of the twenty-four examined, twenty-one, or 87.5 per cent, had one or more of the signs described under the syndrome of myelodysplasia and that six of ten examined roentgenographically showed vertebral anomalies. Peritz [15] examined twenty-two adults and twenty children

10. Altshul, A.: Occult Spina Bifida, Med. Klin. **20**:1567, 1924.

11. Virchow, R.: Ztschr. f. Ethnol. **7**:279, 1875.

12. Fuchs, A.: Ueber den klinischen Nachweis kongenitaler Defectbildungen in den unteren Rückenmarksabschnitten (Myelodysplasie), Wien. med. Wchnschr. **59**:2141, 1909.

13. Riley, H. A.: Syringomyelia or Myelodysplasia, J. Nerv. & Ment. Dis. **72**:1, 1930.

14. Mattauschek, E.: Ueber Enuresis, Wien. med. Wchnschr. **59**:2153, 1909.

15. Peritz, G.: Enuresis nocturna und Spina bifida occulta (Myelodysplasie), Deutsche med. Wchnschr. **27**:1256, 1911.

who suffered from enuresis and stated that 68 per cent of the adults and 35 per cent of the children were found to have spina bifida occulta. Hoffman [16] examined one hundred and twenty-five children with enuresis. He found that seventy-four children, or 59.2 per cent, showed spina bifida occulta, thirty-four children showed no evidence of it, while in seventeen the indications were doubtful. Arnesen [17] examined twenty-eight children with enuresis and found spina bifida occulta in 53.6 per cent. Kohler [18] stated that spina bifida occulta can be seen by x-ray in 60 per cent of cases of enuresis.

As mentioned early in this article, every child with enuresis coming to the child guidance clinic of the fifth division of the Neurological Institute, in addition to receiving a complete neurologic examination, was referred, as a routine, for a roentgen examination of the lumbosacral region with the object of demonstrating the presence or absence of spina bifida occulta. Twenty-five children with the complaint of enuresis have been examined. In none of these children were there any changes in the skin over the lumbosacral region. They were also free from sensory or motor disturbances, as evidenced by the routine neurologic examination in the clinic. The youngest of these children was 3 years of age, the oldest was 14 years. Of the twenty-five children, twenty-one, or 84 per cent, showed spina bifida occulta in the lumbosacral region by x-ray picture. The defect in all these twenty-one cases was a cleft or a gap in the neural arch. The location of the defect was as follows: in seven cases, the first sacral segment; in three, the first and second sacral segments; in one, the first sacral and all below the second sacral segment, the second sacral segment appearing normal; in one, the first three sacral segments; in three, the entire sacrum; in one, the fifth lumbar; in three, the fifth lumbar and first sacral, and in two, the fifth lumbar segment and the entire sacrum. As mentioned before, all these children had the complaint of enuresis nocturna; one, aged 10 years, showed the defect in the fifth lumbar segment and the entire sacrum, and this child had had involuntary stools and urine since infancy.

Before drawing any conclusions from what appears to be a relatively high percentage of spina bifida occulta in children with enuresis it is important to see what is the incidence of spina bifida occulta in general. While general statements have been made as to its frequency no accurate data have been published.

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16. Hoffman, W.: Ueber den Röntgenbefund bei Enuresis nocturna (Spina bifida occulta), Fortschr. a. d. Geb. d. Röntgenstrahlen **26**:322, 1919.

17. Arnesen, A. J.: Enuresis and Spina bifida, Norsk mag. f. lægevidensk. **85**:299, 1924.

18. Kohler, A.: Röntgenology, New York, William Wood & Company, 1928.

In 1885, the Committee on Spina Bifida of the London Clinical Society [19] reported as follows: "Dr. Demme found 57 cases of spina bifida in 36,148 children; Chaussier records 22 cases among 22,293 children in the Paris Maternité." Harrar,[20] in 1916, found fifty-nine cases of spina bifida in 91,600 births in the Lying-in Hospital in New York. These figures give an incidence of one in 900 births. These figures omit the cases of spina bifida occulta. Joseph [6] stated that spina bifida with tumor occurred once in 1,000 births, and the frequency is probably greater for spina bifida occulta. Clark [21] stated that of all varieties in all regions spina bifida occulta comprises only 5 per cent, but undoubtedly he referred to cases in which definite neurologic symptoms were shown. Keiller,[3] while giving no figures, stated that cases of spina bifida occulta without symptoms of pressure on the cord are fairly frequently encountered in the dissecting room. From the roentgenologic point of view the fact is recognized that among the most common congenital anomalies of the vertebrae as revealed by the x-ray picture is the nonfusion of the laminae or spinous processes of the fifth lumbar or first sacral segment, giving the effect of linear or transverse fissures. No definite figures are given.

With these general facts as regards spina bifida occulta in mind, arrangements were made with the Babies' Hospital and the pediatrics department of the Vanderbilt Clinic to refer fifty children to the x-ray department as control patients. These control children were entirely free from enuresis as well as from any neurologic symptoms and came to the clinic for some minor complaint such as an infection of the upper respiratory tract. All these children were referred to the x-ray department for an x-ray picture of the spine of the lumbosacral region to determine the presence or absence of spina bifida occulta. Of these fifty controls twenty-seven, or 54 per cent, were reported as showing spina bifida occulta. The defect, as in the series of children with enuresis, was a cleft or a gap in the neural arch. The location of the defect was as follows: in seventeen cases, the first sacral segment; in two, the first and second sacral segment; in five, the fifth lumbar segment; in three, the fifth and first sacral segments, making a total of twenty-seven cases. Twenty-three cases were reported as negative for spina bifida occulta.

The presence of spina bifida occulta in 54 per cent of normal children corresponds very closely with what has been reported concerning the percentage of spina bifida occulta in children with enuresis. These findings certainly do not warrant the conclusion that there is a definite correlation between enuresis and spina bifida occulta. Even in the

19. Tr. Clin. Soc., London **18**:339, 1885.

20. Harrar, J. A.: Bull. Lying-In Hosp. New York **10**:143, 1915-1916.

21. Clark, S. N.: Spina Bifida Occulta in Cervical Region, J. Nerv. & Ment. Dis. **48**:201, 1918.

present series the finding of a somewhat higher percentage of spina bifida occulta in children with enuresis (84 per cent) does not warrant any such conclusion, since the series is rather small.

When, however, one compares the location and severity of spina bifida occulta in the children with and without enuresis one sees that in the children with enuresis the location of the defects is more variable and the defects more extensive. In the series with enuresis were some children showing involvement of the entire sacrum or of the fifth lumbar segment and the entire sacrum, while none of those in the normal series with spina bifida occulta showed any such extensive defects, in spite of the fact that the number of children in the control series was twice that in the series with enuresis.

It is believed that the following inferences can be drawn: First, spina bifida occulta is an indication that abnormal development has taken place in that region. In the majority of cases, however, the abnormal changes in the medullary plate have been so slight and so transitory as to leave no effect on the spinal cord, though leaving a defect in the vertebral column. Second, spina bifida occulta can be considered as an organic basis for enuresis only in those cases which show an extensive involvement of the vertebrae in the lumbosacral region, such as that of the entire sacrum or that of the fifth lumbar segment and the entire sacrum. Spina bifida occulta may also be considered as an organic basis for enuresis in those cases which, in addition to a mild defect of the vertebrae, show some sensory changes of the lower extremities or some other signs such as those previously described.

The most common type of enuresis in children is the nocturnal. During infancy the spinal and vegetative centers are in control of micturition and it is a purely reflex function. As the cortical centers develop, the child gradually learns to control the act of micturition. Control during the day is usually acquired before nocturnal control. When a child more than 3 years of age continues bed wetting while the cause may be a reduction in intensity of cortical control, one may also assume that at night when the activity of the cortical centers is in abeyance any abnormality in the spinal cord will assert itself.

Two theories may be advanced to support the view that there is a correlation between enuresis and spina bifida occulta in the lumbosacral region. The first theory predicates that there is a defect in the lumbosacral roots or in the spinal cord, possibly a myelodysplasia of the conus medullaris. The second theory explains the occurrence of the symptoms as caused by the presence of bands and adhesions pressing on the spinal cord or on the cauda equina. In support of the first theory there is the fact that in frank cases of spina bifida weakness of the bladder is usually present. Wechsler [22] stated that in spina bifida there is usually

22. Wechsler, I. S.: A Text-Book of Clinical Neurology, Philadelphia, W. B. Saunders Company, 1928.

evidence of lumbosacral root degeneration and that the picture may resemble a lesion of the cauda equina. Among the symptoms, he stated, there is incontinence of urine. One may assume that the difference between spina bifida and spina bifida occulta is one of degree, and because of the milder defect the symptoms are not so pronounced. From the experiences of the late war, Jelliffe and White [23] stated that diseases of the conus medullaris or cauda equina may cause disturbances of the bladder. There is no autopsy material to prove the presence of myelodysplasia in cases of enuresis, but among the symptoms that characterize myelodysplasia, enuresis and spina bifida occulta appear in a large proportion of the cases. The other theory, that the symptoms are due to pressure on the spinal cord or cauda equina, has been substantiated in several instances. In patients who have been operated on for spina bifida occulta, bands and adhesions have been found between the abnormally developed bony structures and the conus or cauda equina (Brickner,[24] Jones [25]).

## SUMMARY

1. Spina bifida occulta is a developmental defect which indicates some abnormality in the process of embryogenesis in the region affected.

2. The roentgen observations are frequently the only diagnostic evidence of spina bifida occulta.

3. Fifty-four per cent of normal children show roentgenologically the presence of spina bifida occulta in the lumbosacral region.

4. Spina bifida occulta may be considered as an organic basis of enuresis only in those cases which show an extensive involvement of the vertebrae in the lumbosacral region or which, in addition to a mild defect of a vertebra, present one or more confirmatory signs as described under the symptom complex of myelodysplasia.

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23. Jelliffe, S. E., and White, W. A.: Diseases of the Nervous System, Philadelphia, Lea & Febiger, 1923.

24. Brickner, W. M.: Spina Bifida Occulta, Am. J. M. Sc. **155**:473, 1918.

25. Jones: Spina Bifida Occulta, Brit. M. J. **1**:173, 1891.

# Case Reports

## NEUROCYTOMA DERIVED FROM A GANGLIONEUROMA OF THE HYPOGASTRIC PLEXUS

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CHICAGO

Virchow, in 1864, was the first to recognize that tumors of the suprarenal medulla which metastasize to the liver are neurogenic and to refer to them as gliomas. Marchand,[1] in 1881, following the line of thought initiated by Virchow, reported suprarenal tumor in a 9 year old child and called it a glioma. In 1907, Marchand [2] reported the first authentic case on record of a neurocytoma of the gasserian ganglion. Since then, tumors of the neurogenic type have been described from time to time.

They are known to occur in any place where nerve tissue is present, as in the brain, the retina, the gasserian ganglion, the spinal cord, the abdominal sympathetic system and the suprarenal medulla.

Pepper,[3] in 1901, described a tumor of the suprarenal medulla which he believed to be a lymphosarcoma. The growth, he thought, appeared soon after birth and gave rise to diffuse hepatic infiltrations. This type of tumor is now known as the Pepper type of neurocytoma. It is difficult to differentiate between a lymphosarcoma and a neurogenic tumor. Hutchison,[4] in 1907, described a type of suprarenal sarcoma which was characterized by metastases to bone, particularly to the skull, causing proptosis of one or both eyes. Metastases to the liver may also exist. This group of symptoms carries the name of Hutchison type. There seems to be an abundance of literature on tumors of the suprarenal medulla, but a comparative scarcity on tumors of the hypogastric plexus.

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From the Departments of Pediatrics and Pathology of the Cook County Hospital and the University of Illinois College of Medicine.

1. Marchand, F.: Ueber eine eigenthümliche Erkrankung des Sympathikus der Nebennieren und der peripherischen Nerven, Virchows Arch. f. path. Anat. **81**:477, 1880.

2. Marchand, F., in Festschrift für Georg Eduard von Rindfleisch, Leipzig, Wilhelm Engelmann, 1907.

3. Pepper, W.: A Study of Congenital Sarcoma of the Liver and Supra-Renal, Am. J. M. Sc. **121**:287, 1901.

4. Hutchison, R.: On Suprarenal Sarcoma in Children with Metastases in the Skull, Quart. J. Med. **1**:33, 1907.

Scott, Oliver and Oliver,[5] in an excellent review of the literature, termed ganglioneuroma the benign form of neurocytoma.

## REPORT OF A CASE

*History.*—R. J., a Negro boy, aged 3 years, was admitted to the Cook County Hospital on Aug. 15, 1933, with a diagnosis from the examining room of acute rheumatic fever.

The complaint on admission was swollen joints in the elbows, knees and ankles of three months' duration. The mother stated that the child's head was becoming enlarged. During May the child was ill with rheumatism which affected several joints, leaving one and involving another. The pains were aggravated by the weather, and during the two days prior to admission to the hospital they had grown worse.

In the preceding week the mother had noticed prominence of the veins of the scalp, which seemed to pulsate.

The child's appetite was poor, and he was constipated. There was no loss of weight. At one time he had edema of the ankles. The child had rickets with convulsions during infancy; otherwise the history was essentially unimportant. He was a normal baby born at full term. He had his first tooth at 7 months and walked at 13 months. The father and mother were both living and well.

*Physical Examination.*—A Negro boy, about 3 years old, appeared to be fairly well nourished and not acutely ill, although the temperature was 102.6 F., the respiratory rate, 48, and the pulse rate, 155. The blood pressure was 96 systolic and 56 diastolic. The head was greatly enlarged, with prominent frontal bosses. The coronal and parietal sutures were widely separated. The fontanels were closed. The veins of the scalp were greatly dilated and distended and pulsated considerably. There was a soft swelling about the size of a walnut in the right occipitoparietal region which seemed to move with the scalp when palpated. It appeared to be fluctuant, but not tender. In the left frontal region was a bony swelling, also the size of a walnut, which was not tender. The right eye was slightly bulging but otherwise normal. The teeth were in good condition. The gums and buccal mucosa were pale, and on the right side of the lower jaw behind the first premolar tooth was a soft, spongy, light purple mass covered with a thin layer of inspissated pus. The cervical nodes were the size of a plum and were firm, discrete and movable. There were marked venous pulsations in the neck.

In the chest were diffuse precordial pulsations. There was a palpable systolic thrill over the fourth and fifth interspaces; however, there was no apparent cardiac enlargement. A loud, blowing systolic murmur was heard over the entire precordium, which was transmitted to the axillae and the vessels of the neck. The lungs were essentially normal.

The abdomen was distended, and the umbilicus was bulging. A mass could be seen in the region of the umbilicus and to the left. On palpation a mass could be felt in the left upper quadrant which extended into the pelvis; it was round and about the size of a small grapefruit. The inferior or lower part of the tumor could not be palpated. The mass was easily movable, and did not appear to be tender. The liver was palpable two and a half fingerbreadths below the costal margin, and the edge was sharp. The spleen could not be felt. The inguinal

5. Scott, E.; Oliver, M. G., and Oliver, M. H.: Sympathetic Tumors of Adrenal Medulla, Am. J. Cancer **17**:396 (Feb.) 1933.

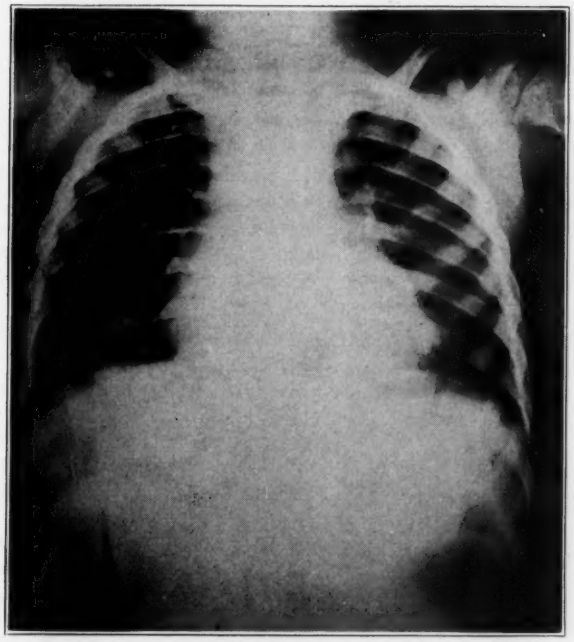

Fig. 1.—Subperiosteal metastases to the ribs. Note the uniform thickening of the periosteum.

Fig. 2.—Sclerosis with areas of destruction in the lower third of the right femur and upper part of the tibia. Periosteal thickening of the femur is evident.

glands were markedly enlarged, being the size of a walnut. They were fully movable and not tender.

The right knee joint was moderately swollen and tender. The swelling extended two thirds of the way up the anterior part of the thigh. Movement of the joint did not cause pain. The other joints seemed normal. There was marked tenderness on pressure over both tibias. The epitrochlear glands were the size

Fig. 3.—Periostitis of the femur, tibia and fibula.

of a peanut and readily visible. On rectal examination a mass could be felt, which was movable, round and not tender. The reflexes were normal. There was no reaction to the various tuberculin tests.

On entrance, the hemoglobin content was 35 per cent; the red blood cell count was 1,000,000, and the white blood cell count was 9,500, with polymorphonuclears, 68 per cent, lymphocytes, 31 per cent, and monocytes, 1 per cent. The Wassermann and Kahn reactions of the blood were negative. The cerebrospinal fluid

gave negative reactions to the Pandy test and also to the Wassermann and Kahn tests. The urine was normal. The blood chemistry was as follows: urea nitrogen, 20.54 mg. per hundred cubic centimeters; cholesterol, 250 mg., and plasma proteins, 6.28 Gm.

Roentgenograms of various bones (figs. 1, 2, 3 and 4) showed periostitis of the upper third of the left humerus, several ribs, the clavicles, the right femur and the upper portion of the tibia. The bones of the skull showed a granular appearance, with a lack of osseous detail and marked separation of the coronal suture. There was sclerosis, with areas of destruction in the lower third of the right femur and upper part of the tibia. The heart showed moderate enlargement.

*Course.*—The child's temperature varied between 99 and 104 F. On August 18, two days after admission, the abdomen became distended. The heart murmur

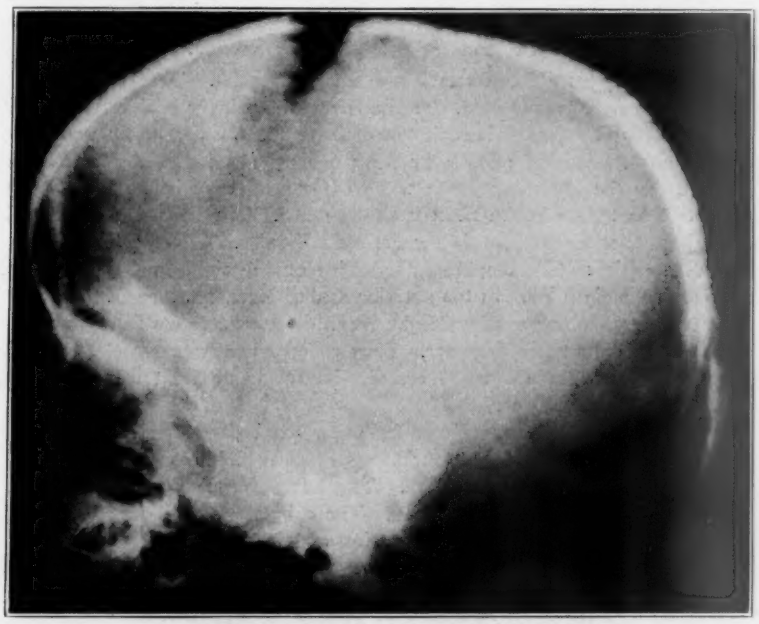

Fig. 4.—Granular appearance of the skull, with lack of osseous detail and marked separation of the coronal suture.

persisted. My associates and I felt that the cardiac condition might be congenital. On August 21 the eyelids became swollen, and dyspnea and orthopnea became marked. Pitting edema of the lower extremities extended up the legs. The eyelids became more edematous and the abdomen more distended, with shifting dulness. The scrotum also became edematous.

Because the heart was not appreciably enlarged and the liver was not easily palpable, it was thought that the tumor might be interfering with the return circulation, causing edema. The child continued to decline, and he died on August 23.

Various diagnoses were considered, including rheumatic heart disease with a neoplasm, and almost all of the malignant tumors—neurocytoma, neuroblastoma, lymphosarcoma and the rest.

The autopsy was performed by Dr. R. H. Jaffé.

*Macroscopic Postmortem Examination.*—External Observations: The body was that of a fairly well nourished, narrowly built Negro boy. There was marked edema of the scrotum. In the region of the right posterior parieto-occipital region was a soft, subcutaneous swelling adherent to the bone, 30 by 30 mm. and 10 mm. high. In the right parietal region was a distended vein running vertically. Similar veins were seen in the right temporal region. There were moderate chemosis of both eyes and slight bulging of the right eye. The mucosa of the lips was light purple-brown and dry. There were eight teeth in the lower jaw and nine in the upper jaw, all in good condition. In the lower jaw on the right, behind the first premolar tooth, was a soft, spongy, light purple mass covered with a thin layer of inspissated pus, measuring 12 by 10 by 6 mm. The cervical nodes were the size of plums, firm, discrete and movable. The maxillary lymph nodes were the size of peanuts, and the inguinal nodes were as large as walnuts. The abdomen was distended, and the umbilicus was bulging.

Abdominal Cavity: There was 2 mm. of midline fat. The cavity contained 200 cc. of clear, light yellow fluid. To the left of the urinary bladder, bulging into the abdominal cavity just below the innominate line of the pelvic bone was a moderately firm, lobulated mass 8 by 8 by 6 cm. The mass was covered with peritoneum. In the upper portion it was dark purple-gray; in the lower portion, pink-gray. The spleen lay in the postaxillary line. The liver was 9 cm. below the xiphoid process and 8.5 cm. below the costal border.

Pleural Cavities: The diaphragm extended on the right to the fifth rib and on the left to the sixth rib. In the anterior mediastinum, attached to the posterior aspect of the sternum, were several light grayish-tan, moderately firm lymph nodes measuring up to 20 mm. in diameter. They covered the large vessels. The right pleural cavity contained about 150 cc. of slightly cloudy light brown fluid, and the left cavity, about 100 cc.

Thymus: The body was replaced by loose connective tissue.

Pericardial Sac: Fifty cubic centimeters of clear fluid was found.

Heart: The weight was 113 Gm. The left ventricle measured 10 mm. in diameter, and the right ventricle, 3 mm. The epicardium was smooth and shiny, and the myocardium was light purplish pink and friable. The left ventricle was slightly dilated, measuring 6 cm. in both the transverse and the vertical diameter.

Aorta: The aorta measured 42 mm. in diameter, and there were fatty plaques.

Arteries: The pulmonary artery was 41 mm. in diameter; its intima as well as that of the coronary arteries was smooth.

Lungs: The lungs were moderately distended and subcrepitant, with a surface of light purple-gray mottled with darker purple-gray. On sectioning they were crepitant and pale gray. The basal portions of the lower lobes were darker purple. The mucosa of the bronchi was pale. The bifurcation lymph nodes, 30 by 15 by 10 mm., were soft, moist and purple-red.

Thyroid: The gland, weighing 4 Gm., was light purple-brown and of uniform consistency.

Stomach: The mucosa was light gray-white; the folds were distinct.

Gallbladder: This was distended and filled with golden yellow, thin bile.

Pancreas: This weighed 80 Gm. and was lobulated and firm. The head was surrounded by a mass, 40 by 30 by 30 mm., of firm, light grayish-brown to grayish-white lymph nodes which bulged deeply into the parenchyma. Similar nodes were found about the body and the tail.

Intestines: The mucosa of the small intestine was light gray-brown and pale, and that of the large intestine was thickened and injected, from purple-brown to purple-gray, with purple-red patches the size of a pinhead.

Suprarenal Glands: Each body weighed 8 Gm. The cortex, which was light orange-yellow, was 1 mm. thick.

Kidneys: Each kidney weighed 170 Gm. The consistency was diminished, and the capsule stripped easily. The surface was smooth and light gray. The cortex measured 10 mm. in thickness and was light purple-brown and moist; the markings were indistinct. The pelvis of the left kidney was moderately dilated; the mucosa was pale.

Pelvic Organs: The urinary bladder was filled with clear, light yellow urine. The mucosa was pale. The mass to the left of the bladder, previously described, was adherent to the wall of the bladder and slightly compressed the left ureter. On sectioning it was seen to consist of an upper portion, deep purple-red, measuring 6 by 5 cm., and a lower portion, light tan, 8 by 6 cm.

Testicles: Each weighed 3 Gm. and was firm. On sectioning the color was light gray-brown. In the left testicle was a sharply circumscribed, homogeneous, grayish-white node, 10 mm. in diameter, surrounded by a pale purple-red zone.

Lymph Nodes: The iliac and aortic nodes were matted together into firm masses measuring up to 80 by 50 by 40 cm. On sectioning, the masses were light purple-red and mottled with dark purple-red and opaque yellow-gray.

Bone Marrow: That of the shaft of the right femur was light tan-gray and was firm. It contained a firm, light yellow-gray nodule, 4 mm. in diameter.

Skull: The skull measured 17.5 by 15 cm., with an average thickness of 5 mm. At the posterior margin of the right parietal bone was a soft yellow and purple mass, 3 cm. in diameter and 1.5 cm. high (fig. 5). The sutures between the parietal and the frontal and between the occipital bones were loosened enough to separate the bone. The entire epidural space was occupied by a confluent mass of soft nodules (fig. 6) which were mottled light yellow-gray and deep purple and varied from 1 to 2.5 cm. in height. The nodules compressed the brain but did not break through the dura, except for small areas, from 10 to 15 mm., at the tips of the frontal bones. At the most anterior portion of both frontal bones the tumor invaded and penetrated the bones, and formed a firm yellow mass, 3 cm. in diameter and 0.5 cm. in height, over the external table. Elsewhere, tumor nodules were firmly adherent to the inner table of the skull; except for the masses over the frontal bone and the one over the right parietal bone they did not seem to have invaded the bone. At the site of the mass over the right frontal bone, the bone was much softened and broke easily. The inner surface of the dura contained numerous purple areas measuring 2 mm. in diameter. The middle ears were free. The space ordinarily occupied by the sphenoid sinus and the retrobulbar spaces were filled with yellow-gray to purple-gray tumor tissue.

Brain: The brain weighed 1,190 Gm. The leptomeninges were injected, and the convolutions were flattened. The consistency was very soft. The cut surface showed the cortex and central ganglions to be brown-gray. The ventricles were not dilated. The vessels at the base of the brain were thin, and the walls were smooth.

Miscellaneous: The lower half of the right femur was much thickened by subperiosteal, soft, yellow-gray masses of tissue similar to that described. The masses increased the diameter of the bone to 4 cm. in the lower half, whereas it was from 1 to 0.8 cm. in the midportion. The knee joint had no obstruction to motion.

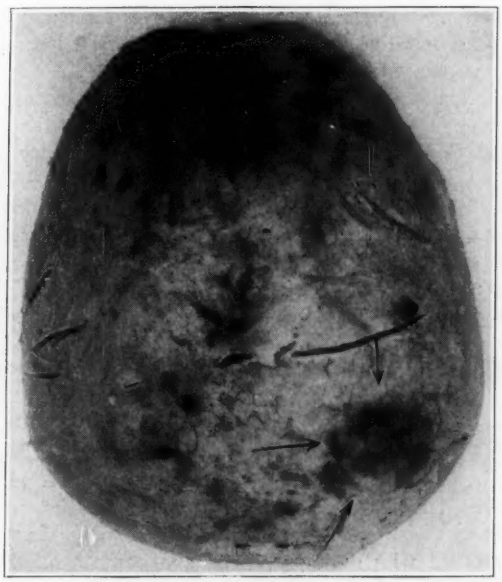

Fig. 5.—A subperiosteal tumor of the skull which was movable because bone was eroded.

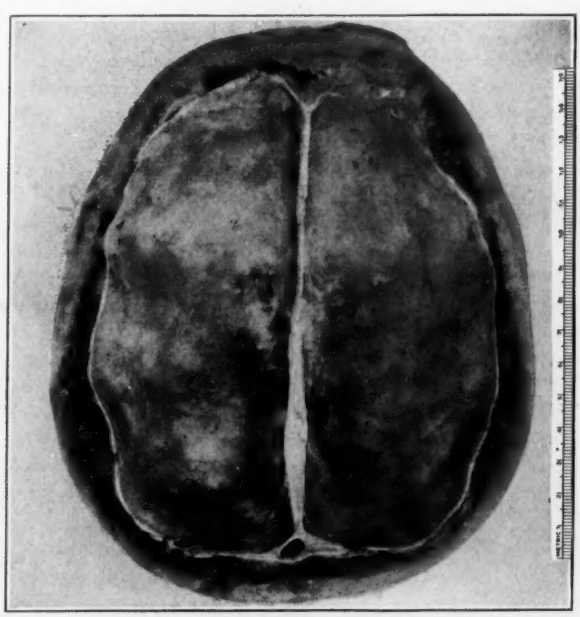

Fig. 6.—Subdural metastases compressing the brain and eroding bone.

*Microscopic Postmortem Examination.*—Pelvic Mass: One part of the mass was composed of interlacing bundles of single fibers, between which were elongated, band-shaped nuclei with fine chromatin granules. The fibers surrounded groups of large ganglion cells, many of which had two nuclei, with prominent nucleoli.

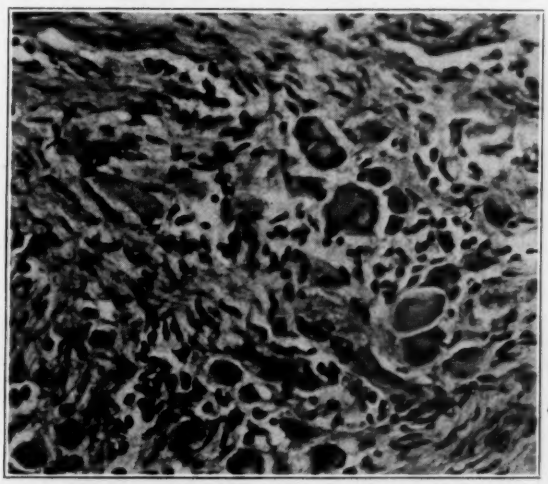

Fig. 7.—Ganglioneuroma of the hypogastric plexus, showing ganglion cells with nerve fibers; × 300.

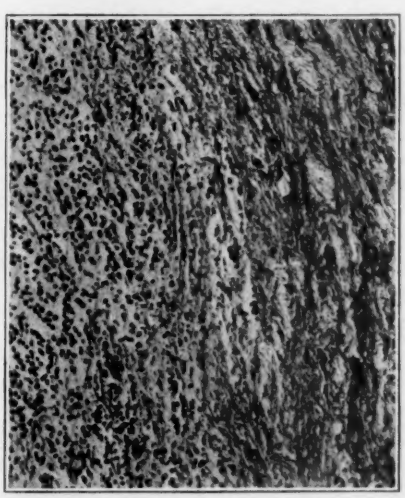

Fig. 8.—Ganglioneuroma and neurocytoma; × 150.

The ganglion cells were surrounded by well developed capsule cells. Some of the ganglion cells showed regressive changes in the form of shrinking, homogenization and pyknosis of the nucleus. A few of the ganglion cells were vacuolated. Scattered between the fibers and ganglion cells were small groups of dark, nucleated round cells slightly larger than lymphocytes. In the periphery

the mass was surrounded by a capsule of dense connective tissue, from which thin septums with blood vessels extended into the underlying tissue. In the capsule, as in the periphery of the mass, were single, typical nerve bundles which appeared to be more compact than the fibrils previously described, from which they were separated by a thin capsule. The dark red portion of the mass revealed an entirely different structure. Separated by extravasations of blood were large alveoli composed of round cells about twice the size of lymphocytes. Many had shrunken and pyknotic nuclei, with an indistinct rim of cytoplasm. The nuclei, which were better preserved, contained a dense net of fine chromatin granules, and the pale-stained cytoplasm was seen extending into fine fibrils which occasionally formed small bundles. At the border of the two structures, small alveoli of round cells were seen extending into the fibrillar portion. The alveoli showed delicate fibrils connected with the cells.

Iliac Lymph Nodes: There were only small islands of lymphatic tissue, while most of the glands were composed of dense cell masses. The masses were formed

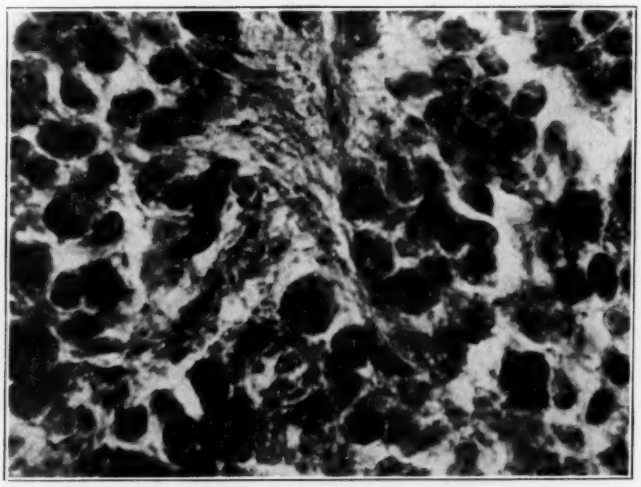

Fig. 9.—Neurocytoma, showing undifferentiated nerve cells with undifferentiated neurofibrils. Note the formation of rosettes; × 1,100.

by cells with a round or slightly oval nucleus rich in chromatin and a pale-stained cytoplasm. The cytoplasm extended into distinct fine fibrils which often were clumped together, forming a central, finely fibrillar mass about which the nuclei were arranged in the form of a rosette. Many cells had shrunken and pyknotic nuclei, and there were many small hemorrhages. The cells were subdivided into irregular alveoli by fine and branched connective tissue septums with dilated blood vessels.

Testicles: The undifferentiated seminiferous tubules were separated by an increased amount of fibrillar connective tissue. In the region of the circumscribed nodule the connective tissue contained islands of dark, nucleated round cells, many of which showed distinct, fine fibrils without relation to the stroma.

Pancreas: There was a large area in which the pancreatic tissue had been replaced by dense accumulations of round cells like those previously described. The production of fibrils by these cells was distinct, and there were occasionally

whole groups of fibrils surrounded by nuclei. The tumor was traversed by many-branched connective tissue septums which contained small and medium-sized, well preserved ducts. The tumor was not sharply separated from the pancreatic parenchyma.

Liver: There was a large nodule as well as several smaller nodules composed of tumor cells. Adjacent to the tumor nodules were small groups of tumor cells lying within the lumens of the portal capillaries. The hepatic cells contained a moderate number of medium-sized droplets of fat.

Dura Mater: Attached to the external aspect of the dura mater was a thick layer of cells with numerous confluent extravasations of blood. In many places, the cells formed rosettes about bundles of fine fibrils.

Mass in the Gum: The surface was covered with a thin layer of necrotic tissue, with clouds of bacteria which rested on a cellular granulation tissue. Underneath the granulation tissue and extending into its basal part were groups of closely packed, dark-nucleated round cells, which often seemed to be located in preformed spaces lined with endothelium.

Mass in the Femur: Underneath the periosteum was a thick layer of tumor tissue, into which perpendicular septums of connective tissue extended. The cells often possessed fine fibrils, but there were no distinct rosettes. The innermost portion of the periosteum showed parallel, palisade-like layers of tumor cells.

Bone Marrow: This contained numerous nodules of densely packed tumor cells. The stroma between the cell masses revealed numerous mature oxyphilic leukocytes and a few neutrophilic myelocytes. About the nodules the bone marrow was fibrotic.

Spleen: The pulp was congested, and in the cords were many plasma cells, lymphoid round cells and oxyphilic leukocytes and myelocytes. There was a small amount of brownish pigment in the reticular cells. The follicles were large and lymphatic, with an outer zone of larger lymphoid round cells.

Peritracheal Lymph Nodes: The nodes were partially replaced by tumor tissue. In the intact portions the sinuses were dilated and filled with red blood cells.

Spleen: The organ weighed 105 Gm. and measured 11 by 7 by 4.5 cm. The capsule was covered with a thin, loosely adherent, light brown membrane. It was of firm consistency. A sectioned surface revealed many follicles the size of a pinhead.

Liver: The organ weighed 1,630 Gm.; it was moderately firm, and the surface was light purple-brown and smooth. A sectioned surface was light brown-gray, without structural markings. On the inferior border of the left lobe near the hilus was a gray-white, centrally umbilicated nodule, 9 mm. in diameter. Near the nodule was a similar one, 2 mm. in diameter. On the anterior aspect of the right lobe were two nodules measuring 2 mm. and one measuring 3 mm.

*Anatomic Diagnosis.*—Included in the diagnosis were: neurocytoma derived from a ganglioneuroma of the hypogastric plexus; metastases to the left testis, both femurs and tibias, the bone marrow, the peri-iliac, periaortic, peritracheal and posterior mediastinal lymph nodes, the right mandible and gum, the epidural space of the skull, both frontal bones and the right parietal bone, the ethmoid and sphenoid sinuses, the retrobulbar tissue of both eyes and the pancreas and liver; marked compression of the brain; separation of the coronal and occipital sutures by pressure; parenchymatous degeneration of the myocardium, and inflammatory swelling of the spleen.

## COMMENT

This case is interesting because it shows the nonmalignant form of neurocytoma in the form of a ganglioneuroma, and also the true neurocytoma in the same section.

An unusual form of metastasis to bone is shown in which the periosteum is uniformly thickened.

## SUMMARY

A case of ganglioneuroma of the hypogastric plexus is reported in which microscopic section showed the transformation into the malignant form, neurocytoma.

# EXTERNAL HYDROCEPHALUS DUE TO STENOSIS OF THE LONGITUDINAL SINUS AND INTERNAL HEMORRHAGIC PACHYMENINGITIS

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OGDEN, UTAH

External hydrocephalus has received scant mention in the medical literature of the past decade which contains so much of value concerning the nature and causes of the internal form. Blackfan [1] even questions the advisability of considering it a clinical entity since its pathogenesis is so imperfectly understood. He said: "It is an exceedingly rare condition closely associated with and in all probability secondary to internal hydrocephalus." During recent years, however, several papers have appeared dealing with internal hemorrhagic pachymeningitis and chronic subdural hematoma in which mention is made of the presence of considerable quantities of yellow or bloody fluid in the subdural space with a widening of the cranial sutures and enlargement of the head. Penfield [2] described a case in which there was moderate dilatation of the ventricles and also a large collection of fluid in the subdural space but no communication between the subdural and subarachnoid spaces. The subdural fluid was yellow, contained a high amount of protein, with no evidence of blood, and was thought to be of inflammatory origin.

Burhans and Gerstenberger [3] described five cases of internal hemorrhagic pachymeningitis, four of which were probably of traumatic origin. Noticeable enlargement of the head was not present in any of these infants, although in one case 200 cc. of bloody fluid was withdrawn at one time by fontanel puncture. Hunt [4] reported three cases of hydrocephalus occurring after internal hemorrhagic pachymeningitis among five survivors in a series of six cases. The fluid was probably subdural in these cases, since considerable quantities were obtained by fontanel puncture during the acute stage of the disease. Moderate enlargement of the head of the hydrocephalic type was present in most of the nine infants with subdural hematoma whose cases were reported by Peet

1. Blackfan, K. D., in Abt, I.: Pediatrics, Philadelphia, W. B. Saunders Company, 1925, vol. 7, p. 398.

2. Penfield, W. G.: Subdural Effusion and Internal Hydrocephalus, Am. J. Dis. Child. **26**:382 (Oct.) 1923.

3. Burhans, C. W., and Gerstenberger, H. J.: Internal Hemorrhagic Pachymeningitis in Infancy, J. A. M. A. **80**:604 (March 3) 1923.

4. Hunt, Fred C.: Internal Hemorrhagic Pachymeningitis in Young Children: A Report of Six Cases, Am. J. Dis. Child. **39**:84 (Jan.) 1930.

and Kahn.[5] The color of the subdural fluid ranged from orange or amber to bloody. All of these patients were treated surgically. Schwartz [6] reports a case with meningeal symptoms and bloody spinal fluid from which a gram-negative diplococcus was repeatedly isolated. Symptoms of hydrocephalus developed, and after about three months amber-colored fluid was obtained from the subdural space. Spinal fluid taken at the same time was perfectly clear. For a full discussion of chronic subdural hematoma, the monograph of Sherwood [7] should be consulted. He reports nine cases in six of which the hemorrhage was bilateral. Hydrocephalus was shown by roentgen examination in four of five cases examined. The head was enlarged in six cases, slightly enlarged in two, and not enlarged in one instance.[8]

The case here reported was unquestionably a pure form of external hydrocephalus, unassociated with the internal type, in which a reasonable explanation of its genesis is possible in the light of the history and autopsy observations.

## REPORT OF A CASE

*History.*—A female infant, 6 months of age, was admitted to the hospital in a comatose condition on the sixth day of her illness. She was an adopted baby, hence her antecedents could not be fully investigated. The mother, however, was a healthy young primipara, whose labor was normal. One dose of morphine, ⅙ grain (0.0108 Gm.), and scopolamine, 1/200 grain (0.00033 Gm.), was given three hours before delivery; gas was given during second-stage pains. No difficulty was experienced in resuscitating the baby. She was taken from the hospital by her foster parents on the fourth day after birth, having gained weight and apparently being in normal condition. During the first two months of her life she made fairly normal progress, doubling her weight at birth. She was always nervous, however, slept very lightly, and was easily aroused. After two months the nervous symptoms became more pronounced. She fretted a great deal at night and would lie whimpering for long periods. She had vomited frequently from the first, and this symptom became more pronounced after about three months. The foster mother felt that the child's intelligence was normal for her age. It was noted, nevertheless, that she showed very little interest in her toys and had made no effort to sit up by the time she was 6 months of age. About six days before her admission to the hospital the restlessness became more pronounced. She refused her feedings and finally lapsed into a state of semiconsciousness.

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5. Peet, Max M., and Kahn, Edgar A.: Subdural Hematoma in Infants, J. A. M. A. **98**:1851 (May 28) 1932.

6. Schwartz, A. B.: The Etiology of Pachymeningitis Hemorrhagica Interna in Infants, Am. J. Dis. Child. **11**:23 (Jan.) 1916.

7. Sherwood, David: Chronic Subdural Hematoma in Infants, Am J. Dis. Child. **39**:980 (May) 1930.

8. Neff, Frank C.: Internal Hemorrhagic Pachymeningitis of Infancy, in Abt, I.: Pediatrics, Philadelphia, W. B. Saunders Company, 1925, vol. 7, p. 559. (A brief but comprehensive chapter on this condition.)

*Examination.*—Examination at the hospital showed a very sick, dehydrated infant with a temperature of 100 F. The significant physical findings were: weakness and drooping of the right side of the face, widening of the left palpebral fissure, twitching of the right upper extremity (less marked of the right lower extremity) and normal patellar reflexes.

*Course.*—During the first week of hospitalization she had frequent convulsive seizures, involving sometimes one side of the body and sometimes the other, with occasional attacks of general rigidity. Deep stupor frequently developed between periods of excitation. By the fifth day her temperature gradually had risen to 106 F., but on discharge it had declined and was normal.

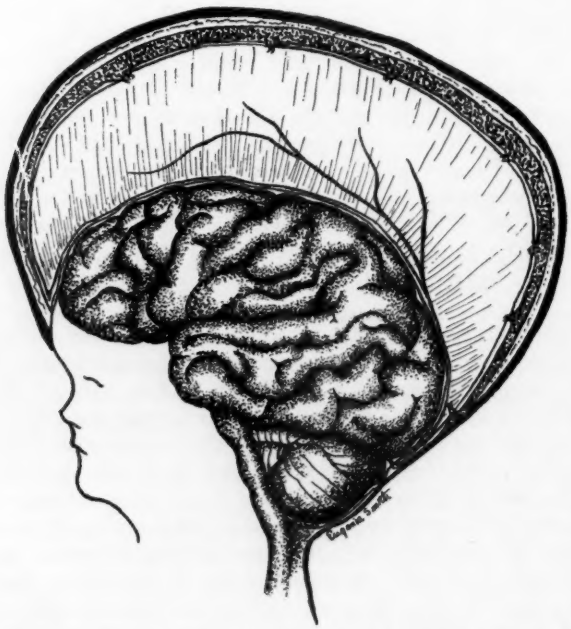
A lumbar puncture was done at home on the day of admission, and about 90 cc. of very bloody fluid was obtained. Later on the same day fluid of like character but under less pressure was removed by a spinal puncture. The fontanel at this time was tense, but the spinal fluid pressure appeared to be about normal. On the fourth day the spinal fluid was still bloody, but only a small amount was obtained. On the seventh day the spinal fluid was clear and contained only a few red blood cells. Smears and culture of the spinal fluid failed to show any organisms. Blood examination at the time of admission showed: coagulation time, three minutes; hemoglobin, 45 per cent (Dare); white blood cells, 11,800; red blood cells, 2,400,000. The white cell count reached 25,200 on the fourth day and 12,400 on the twelfth day. The platelets numbered 326,000. The Wassermann test of the blood was negative, and the urine was normal during her stay in the hospital.

Retinal hemorrhages were noted in both eyes on the day of admission. On the day of discharge ophthalmoscopic examination revealed bilateral hemorrhagic retinitis and considerable papilledema of the right disk. The details of the left disk were obscured by massive hemorrhage, covering the upper half.

For a short time after leaving the hospital the baby seemed to do well, except that she vomited rather frequently. About two weeks after her return home it was noted that her head was increasing in size. When she first came under my care at the age of 7 months, two months after the onset of acute symptoms, she had a marked hydrocephalus with tense and bulging fontanels. Her head was 48 cm. in circumference. She was greatly emaciated, and her weight was 5.6 Kg. (12 pounds and 5 ounces). She was restless and fretful, took her food poorly, and vomited a great deal. Several punctures were made through the anterior fontanel to relieve pressure. Clear, straw-colored fluid was encountered immediately beneath the dura. As much as 120 cc. was removed at one time. Leakage through the puncture wound occasionally continued for several hours. For a day or two following the puncture there was slight relief of pressure symptoms, but the fluid rapidly reaccumulated. She died of exhaustion when 11 months of age, five months after the onset of acute symptoms.

*Autopsy.*—A postmortem examination of the head only was made twelve hours after death. The body was extremely emaciated, and the head greatly enlarged. Both fontanels and the cranial sutures were wide and tense and partially bridged over by parchment-like, new bone formation. Immediately under the dura a large amount of clear, yellow fluid was encountered. The brain occupied the bottom of the enlarged cranial cavity and retained practically its normal size and contour. The right cerebral hemisphere was somewhat smaller than the left. The dura was smooth and much more dense than normal. The falx was thickened and divided the space above the hemispheres into two compartments containing the fluid. That portion of the falx above the level of the hemispheres varied in depth

from 1 to 4.5 cm. The temporal lobes did not extend to the bottom of the middle cerebral fossae but rested on a membrane which divided the latter into an upper and lower compartment. This membrane was thin and was pierced by several round openings, from 0.5 to 1 cm. in diameter. Over each hemisphere the fluid was enclosed in a cystlike sac, adherent to the under surface of the dura above and slightly less adherent to the arachnoid below. This sac did not dip into the median fissure but extended across to the falx at the level of the top of the hemispheres to become continuous with the membrane lying just beneath the dura. The portion of the membrane attached to the dura was somewhat thicker than that overlying the arachnoid. The inner surface of these cavities was smooth and



Semidiagrammatic drawing showing the greatly enlarged subdural space and the position of the brain in the cranial cavity.

free from adhesions. The lumen of the longitudinal sinus was larger than normal in its anterior portion, but became narrowed posteriorly until it became obliterated about 5 cm. above its entrance into the lateral sinus. It contained no blood. The straight and lateral sinuses were patent. When the hemispheres were lifted anteriorly, numerous adhesions were encountered in the region of the olfactory bulb, and several rather large veins were noted communicating with the longitudinal sinus in this region. Underneath the cystic membrane the pia and arachnoid could easily be identified and appeared normal. As stated, the hemispheres were approximately normal in contour. The convolutions were somewhat flattened. The cerebellum was normal in size as compared with the cerebrum and somewhat flattened from above downward. On section the gross appearance of the brain was normal. The lateral ventricles were not enlarged.

COMMENT

There is little reason to doubt that preceding the development of the hydrocephalus the patient suffered an acute subdural hemorrhage. Furthermore, the presence of a preexisting intracranial defect may be surmised from the history of fretfulness, vomiting, and retarded mental development over a period of several months prior to the onset of acute symptoms. This assumption was confirmed at autopsy which disclosed the presence of a stenosis of the posterior portion of the longitudinal sinus.

It appears most probable that the stenosis of the sinus in this case was congenital. Sections of the sinus walls failed to show any evidence of inflammation or of a preexisting thrombus. The lumen of the vessel was filled with a proliferation of the endothelium from the intima but this was not of the tumor type.[9] Obstruction of the longitudinal sinus, even when complete and associated with that of the lateral sinuses, has no effect on the absorption of the spinal fluid from the subarachnoid space and does not produce a hydrocephalus.[10] Byers and Hass [11] in a study of fifty cases of thrombosis of the intracranial venous sinuses occurring in infancy and childhood found no instance in which hydrocephalus occurred as a sequel of uncomplicated thrombosis. They suggest, however, that unrecognized thrombosis may have an etiologic relationship to certain cases of hydrocephalus as well as to other puzzling intracranial conditions. In this case the chronic congestion, resulting from obstruction of the longitudinal sinus, would easily account for the nervous symptoms from which the infant suffered. This congestion was no doubt also an important predisposing factor in the genesis of the subdural hemorrhage. In reported cases occurring in otherwise normal persons it has been noted that such hemorrhage frequently follows comparatively slight trauma. With engorged veins, which undoubtedly existed in this case, very moderate injury would be sufficient to cause such an accident. The point of origin of the hemorrhage as noted by other observers is also that which may be reasonably assumed in this case, namely, from veins extending from the cortex across the subdural space to the longitudinal sinus.

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9. Through the kindness of Dr. A. W. Adson of the Mayo Clinic, Dr. Kernohan, neuropathologist, examined a section of the longitudinal sinus from a point near the site of obstruction. He reported: "The longitudinal sinus is filled with a proliferation of endothelium from the intima but this is not of the tumor type. There are two small channels in this mass which suggests that some blood has passed through, but the total volume has undoubtedly been reduced to a limited quantity."

10. Hassin, George B.: Abscess and Thrombosis of the Superior Longitudinal Sinus, Arch. Neurol. & Psychiat. **29**:359 (Feb.) 1933.

11. Byers, R. K., and Hass, G. M.: Thrombosis of the Dural Venous Sinuses in Infancy and in Childhood, Am. J. Dis. Child. **45**:1161 (June) 1933.

As may be noted in the history, the baby seemed to recover from the immediate effects of the intracranial hemorrhage and was fairly well for a short time. In this respect also the development of symptoms in this case corresponds closely with that of cases definitely known to be due to subdural hemorrhage, the result of trauma. In the latter a characteristic feature is a latent period of weeks or months which often follows the injury. At the end of this time, during which the patient may be free from symptoms, or largely so, signs of intracranial pressure develop with great rapidity, due to the accumulation of fluid in the subdural space. The explanation offered by Gardner [12] as to the cause of the latent interval and the formation of membrane and subdural fluid is supported by a considerable clinical experience and by laboratory experiment. His conclusions are as follows:

> Following a cranial trauma, hemorrhage occurs into the subdural space, probably from rupture of one of the cerebral veins where it crosses the space to enter the sagittal sinus. A large amount of blood having escaped, becomes clotted, and within the course of a few days this clot is surrounded by a capsule of mesothelium and connective tissue growing out from the dura. That this capsule forms with remarkable rapidity is attested by clinical as well as by experimental observations. The portion of the capsule next to the dura becomes invaded with nutrient capillaries from the dura and thus attains a greater thickness than does the avascular arachnoid. The encapsulated clot then undergoes partial liquefaction with a resultant fluid high in protein content. This fluid is separated from the cerebrospinal fluid, which is of low protein content, merely by the thickness of a few layers of cells constituting the inner wall of the cyst and the arachnoid membrane. There then results an ideal set-up for osmotic interchange. Since the neomembrane is impermeable to the large protein molecules in the hemorrhagic fluid, an osmotic unbalance in favor of the hemorrhagic fluid must exist, resulting in the withdrawal of cerebrospinal fluid into the cyst. This, of course, causes a progressive enlargement of the hemorrhagic cyst and eventually a rising intracranial pressure.

## CONCLUSIONS

From the history and the autopsy observations the development of hydrocephalus in this case may be stated to have occurred as a result of several factors. A marked narrowing, if not a complete occlusion, of the posterior portion of the longitudinal sinus resulted in chronic engorgement of the veins emptying into it. This circulatory disturbance was the cause of the irritability, vomiting and other nervous disturbances from which the infant suffered during the early months of life. When about 6 months of age, a subdural hemorrhage occurred, probably the result of some unnoticed trauma. She recovered from the immediate effects of this accident and seemed fairly normal (the latent period) until the subdural effusion was augmented by the osmosis of spinal and other fluids producing an external hydrocephalus, which occupied a considerable portion of the greatly enlarged cranial cavity.

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12. Gardner, W. James: Traumatic Subdural Hematoma, with Particular Reference to the Latent Interval, Arch. Neurol. & Psychiat. **27**:847 (April) 1932.

# Progress in Pediatrics

## INFANT FEEDING AND NUTRITION

### A DECADE OF PROGRESS

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Infant feeding—so simple a problem among simple people—has become, with advancing civilization, almost a major science occupying a great portion of the interest of a special field of medicine. In the past decade a vast amount of work has been done on this problem; a bewildering array of articles has been published. I felt that a culling of this vast granary in an effort to discover what is new, of interest or of importance might be of value. Obviously, a résumé of every paper would defeat this purpose. Therefore, individual judgment has been used, but I believe that sufficient references have been appended to assist any reader interested in a special problem.

Although classification is difficult, the work in general falls into these major groups:

Breast milk and feeding of the new-born
Artificial feeding
- *A.* Acidified milk
- *B.* Protein milk
- *C.* Evaporated, dried and condensed milk
- *D.* Concentrated feedings
- *E.* Carbohydrates
- *F.* "Milkless" diets: vegetable milks
- *G.* Special group

Mineral metabolism
Vitamins

### BREAST MILK AND FEEDING OF THE NEW-BORN

It has been said: "A pair of substantial mammary glands have the advantage over the two hemispheres of the most learned professor's brain in the art of compounding a nutritive fluid for infants."

Few will deny that breast milk is still, in the vast majority of instances, the premier and incomparable food for human infants, in spite of the many varied and ingenious methods of complementing, supplementing or supplanting it. Cow's milk has been acidified, diluted,

concentrated, peptonized, evaporated, dried and homogenized; that it still remains a makeshift substitute is attested to by the multitude of methods used to modify it. The modern mother has, however, been rather prone, because of economic or social factors, to neglect breast feeding in spite of the vigorous campaigns that have been waged in its behalf by both public officials and private physicians. Several reports made during the past decade have stressed the increased mortality and morbidity associated with artificial feeding, especially in the earlier months of life.

Huenekens [1] showed that artificially fed babies have a mortality from three to four times greater than that of breast-fed infants. His statistics for the mortality during the first three months of life are shown in table 1.

TABLE 1.—*Mortality Rate per Thousand Infants*

| | Breast-Fed | Artificially Fed |
|---|---|---|
| First month | 16.9 | 54.7 |
| Second month | 5.8 | 24.6 |
| Third month | 3.7 | 21.2 |

McKay's [2] figures are comparable: Artificially fed infants have a mortality from three to five times higher than breast-fed babies; the death rate for partially breast-fed babies is almost double that of exclusively breast-fed babies; and, considering statistics for the first year only, the longer the period of breast feeding, the lower the infant mortality. Richet,[3] in 1927, reported an infant mortality during the first year of life of 12 per cent for breast-fed children and 31 per cent for artificially fed infants.

However, an occasional slight note of contention has arisen. Thus Glazier,[4] from an analysis of data gathered at health units in different sections of Boston, concluded that infant mortality and frequency of infection are more definitely related to economic and hygienic conditions than to the type of feeding—a point which is well taken. Faber and Sutton [5] found that before the fourth month breast-fed infants show

1. Huenekens, E. J.: Breast Feeding from a Public Health Standpoint, Am. J. Pub. Health **14**:391 (May) 1924.

2. McKay, F. L.: Infant Mortality in Relation to Breast Feeding, New York State J. Med. **24**:433 (March 28) 1924.

3. Richet, C.: Considérations sur l'hygiène du lait, Médecine **9**:179, 1927.

4. Glazier, M. M.: Comparing the Breast-Fed and the Bottle-Fed Infant, New England J. Med. **203**:626 (Sept. 25) 1931.

5. Faber, H. K., and Sutton, T. L.: A Statistical Comparison of Breast-Fed and Bottle-Fed Babies During the First Year, Am. J. Dis. Child. **40**:1163 (Dec.) 1930.

a better gain, but that thereafter the artificially fed children increase in weight more rapidly. The number of infections in the breast-fed group studied was as a rule greater, especially after weaning, which would seem to indicate that neither absolute nor relative immunity to infection is increased by breast feeding. Another report on comparative gains in weight in the two classes of infants has been made by Potter,[6] who showed that while the breast-fed group surpasses the artificially fed group in the average gain of weight during the first six months, during the next six months the latter group definitely outstrips the former, as may be seen in table 2.

TABLE 2.—*Average Gain in Weight*

| | First Six Months, Pounds | Second Six Months, Pounds |
|---|---|---|
| Breast fed | 8.10 | 5.8 |
| Artificially fed | 7.12 | 7.4 |

The question as to how long breast feeding may continue was discussed by Pooler,[7] who has done most of his work among the poorer classes in England. He felt that in districts of low economic level, breast feeding may well continue during the second year of life provided it is complemented with solid food, and that it is preferable to weaning if the cow's milk or its handling is not above suspicion. He found the mothers' milk satisfactory even in the second year of nursing, although there was a lowered albumin and mineral content. According to his statistics the breast-fed group showed a slightly higher average weight, even at 18 months of age, than the weaned group.

Regarding the later development of breast-fed and artificially fed children, Hoefer and Hardy [8] wrote an article of interest. They reported a group of 383 children ranging from 7 to 13 years of age. Artificially fed children were, on the whole, both mentally and physically inferior to breast-fed youngsters. The former were more susceptible to diseases of childhood and, on the average, lagged behind in such developmental phenomena as walking and talking. Children exclusively breat fed for more than nine months were mentally the poorest, but if breast fed for from four to nine months they were superior in every way. The lowest percentage of superior intelligence (intelligence quotient 120 or more) was in the artificially fed group.

6. Potter, P. S.: Observations from Well-Baby Conferences, Arch. Pediat. **47**:784 (Dec.) 1930.

7. Pooler, H. W.: The Value of Human Milk During the Second Year After Childbirth, Arch. Pediat. **47**:698 (Oct.) 1930.

8. Hoefer, C., and Hardy, M. C.: Later Development of Breast-Fed and Artificially Fed Infants, J. A. M. A. **92**:615 (Feb. 23) 1929.

A critical survey of these reports leaves one with the impression that breast feeding for at least the first four months of life is distinctly advantageous.

The desirability, and at times the absolute necessity, of breast milk has led certain investigators to devise methods by which it may be kept over long periods of time. Emerson and his associates [9] have been active in this respect. At first attempts were made to preserve the breast milk by drying by means of a simple, modified roller process. It was found that the preserving qualities were improved by first removing the cream. Later a more satisfactory product was obtained by drying the milk at a lower temperature. Babies to whom the reliquefied dried milk was fed gained in weight rather slowly even when additional carbohydrate was provided. When from 1 to 2.5 per cent calcium caseinate was added, the response, measured by gain in weight, was much better. In the second method, milk was dehydrated by steam at a pressure of 15 pounds (6.8 Kg.). The dried milks were found, by experimentation on animals, to retain 80 per cent of their antiscorbutic value when fresh and 40 per cent after two years. Recently, the preservation of breast milk by freezing has occupied the attention of these workers. Thus far, 25 premature infants have been fed reliquefied breast milk which was frozen from one to two hundred and fifty days. Growth and gain in weight have been eminently satisfactory and almost indistinguishable from the development of a control group fed fresh breast milk.

Breast milk has always been the foundation in the feeding of premature infants. As has been frequently demonstrated, however, its chief disadvantage is that its protein content is insufficient at times for proper growth. This has been remedied in several ways. Poole and Cooley [10] added 2 per cent calcium caseinate to the breast milk for all infants weighing less than 1,800 Gm. (those weighing more than 1,800 Gm. were started on formulas of evaporated milk, beginning with a

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9. (*a*) Emerson, P. W.: The Collection and Preservation of Human Milk: Preliminary Report, J. A. M. A. **78**:641 (March 4) 1922; (*b*) Dried Human Milk as a Food for Infants, Am. J. Dis. Child. **30**:769 (Dec.) 1925; (*c*) Preservation of Human Milk: Feeding to Premature Babies of Human Milk Preserved by Freezing, New England J. Med. **209**:893 (Nov. 2) 1933. (*d*) Smith, L. W.: Experimental Feeding of Dried Breast Milk, J. Biol. Chem. **61**:625 (Oct.) 1924. (*e*) Smith, L. W., and Emerson, P. W.: Notes on the Experimental Production of Dried Breast Milk, Boston M. & S. J. **191**:938 (Nov. 13) 1924; (*f*) Dried Human Milk: Its Use in the Feeding of Premature Infants, Am. J. Dis. Child. **31**:1 (Jan.) 1926. (*g*) Emerson, P. W., and Platt, W.: Preservation of Human Milk: Preliminary Note on the Freezing Process, J. Pediat. **2**:472 (April) 1933.

10. Poole, M. W., and Cooley, T. B.: The Care of Premature Infants, J. Pediat. **1**:16 (July) 1932.

dilution of 1:3 plus 7 per cent additional carbohydrate). Hill[11] achieved remarkable results by changing the formulas of premature babies in his service to 3 parts of breast milk plus 1 part of evaporated milk. The average daily gain in weight was increased from $0.366 \pm 0.12$ ounce to $0.766 \pm 0.11$ ounce. It seemed to Thompson[12] that concentrating the breast milk would overcome one of the chief difficulties in the feeding of premature infants, i. e., their ability to take only a small volume of any formula at a time. By dehydrating the breast milk to half its original volume, thus doubling its caloric value, he was able to offer feedings that were both small in volume and high in caloric value. The results were good. Marfan[13] felt that while breast milk was best in the feeding of premature and hypothreptic infants, it was not always obtainable in the amounts desired. A mixed diet of breast milk and buttermilk, containing at least one third breast milk, proved satisfactory for the early months of life, and was markedly effective in lowering the mortality.

Several conflicting reports have come from Germany concerning the effects of boiling breast milk in the feeding of premature infants. Catel and Walltuch[14] stated that when equal energy quotients in the form of either raw or boiled breast milk were given to premature infants, the group fed raw milk made an average daily gain of 25 Gm., while the other group gained only 14.6 Gm. daily. Furthermore, the mortality from infections of the upper respiratory tract and bronchopneumonia was higher in the latter group. Kayser,[15] however, in a similar experiment failed to discover any difference between two groups of premature infants fed, respectively, raw breast milk and breast milk boiled for twenty minutes. Gain in weight, character of the stools and other phenomena were similar in both groups.

The possibility of the sensitization of breast-fed infants to food proteins in the mothers' milk has been stressed by von Meysenbug.[16]

11. Hill, M. C.: Feeding of Premature Infants, Am. J. Dis. Child. **40**:59 (July) 1930.

12. Thompson, J. R.: Breast Milk Concentrated by Dehydration, Arch. Pediat. **42**:303 (May) 1926.

13. Marfan, A. B.: L'alimentation des débiles et des hypothrepsiques à l'hospice des enfants-assistés, Nourrisson **17**:1 (Jan.) 1929.

14. Catel, W., and Walltuch, M.: Ueber den Einfluss roher und sterilisierter Frauenmilch auf das Gedeihen frühgeborener Kinder, Monatschr. f. Kinderh. **40**: 354, 1928.

15. Kayser, M. E.: Parallelversuche mit roher und sterilisierter Frauenmilch an Frühgeburten, Monatschr. f. Kinderh. **51**:199 (Oct.) 1931.

16. von Meysenbug, L.: Sensitization of Breast Fed Infants to Food Proteins in Mothers' Milk, New Orleans M. & S. J. **76**:421 (March) 1924; Feeding During First Year, with Reference to Some of Its Problems, Arch. Pediat. **44**:215 (April) 1927.

Certain foods, such as eggs, shell-fish and oatmeal, are frequent offenders and are responsible for allergic conditions manifested by colic, vomiting, diarrhea or eczema. The appearance of any or all of these symptoms is frequently the cause of weaning, with the excuse that "the milk doesn't agree with the baby." The discovery of the offending substance by means of a careful dietary history or skin testing, and its elimination from the mother's diet will frequently give complete relief to the nursling.

Toverud [17] emphasized symptoms in breast-fed infants resembling somewhat the allergic phenomena and consisting of dyspepsia, abdominal pain or discomfort, colic, restlessness and failure to gain weight. These were definitely related to an insufficient supply of breast milk. Toverud found low blood sugar levels in many of these infants, and the hypoglycemia was undoubtedly a factor in causing hunger contractions and general increased irritability. However, one should not be too prone to feel that the quantity of breast milk is insufficient merely because of the weight after one or two feedings. Gertsley [18] showed that there may be, and are, great variations in the amount of milk taken at the breast at various feedings and even from day to day. Incidentally, he begged that the rationale be considered in demanding that bottle-fed babies take exactly the same volume at every feeding, while the amount ingested by an infant at the breast will vary greatly according to his appetite and inclination. Nims and her associates,[19] in a careful study of three lactating women, confirmed Gerstley's observations, for they found significant variations in the volume of breast milk obtainable at four hour intervals during the day.

The problem of increasing the vitamin D content of breast milk by various means has occupied the attention of several workers during the past decade. Hess and Weinstock [20] studied a group of 28 pregnant women who were given cod liver oil during the last two months of pregnancy. Rickets developed in the children of 15 of these mothers. In experiments with rats, pregnant animals were fed supplemental diets and cod liver oil previous to and during lactation, but rickets was not

17. Toverud, K. U.: Frequent Cause of Dyspepsia in Breast-Fed Infants, Am. J. Dis. Child. **30**:642 (Nov.) 1925.

18. Gerstley, J. R.: Breast Feeding, M. Clin. North America **12**:185 (July) 1928.

19. Nims, B.; Macy, I. G.; Brown, M., and Hunacher, H. A.: Human Milk Studies: IX. Variation in the Composition of Milk at Four Hour Intervals During the Day and Night, Am. J. Dis. Child. **43**:824 (April) 1932.

20. Hess, A. F., and Weinstock, M.: Rickets as Influenced by the Diet of the Mother During Pregnancy and Lactation, J. A. M. A. **83**:1558 (Nov. 15) 1924; Antirachitic Effect of Cod Liver Oil Fed During Period of Pregnancy or Lactation, Am. J. Dis. Child. **27**:1 (Jan.) 1924.

prevented in the young although it was mitigated somewhat. Hess concluded with the observation that the fact that susceptibility to rickets may be induced by inadequacy of the maternal diet does not mean that it is likewise possible to render the young resistant by fortifying the nutrition of the mother in the prenatal period. He felt that rickets in the offspring could not be prevented by improving the mother's diet either previous to pregnancy or during pregnancy and lactation. Greenbaum [21] gave a group of 25 expectant mothers a high mineral and caloric diet during the last three months of pregnancy, and felt that the rickets that developed in their children was milder than in the children used as controls.

In another experiment conducted by Weech [22] on colored patients, mothers were instructed to take 3 teaspoonfuls of cod liver oil daily for three months following delivery. The children of the mothers who followed instructions fairly diligently showed a serum calcium-phosphorus quotient of 40 or more and the signs of rickets by roentgenogram were not as severe as in the children of the mothers who neglected to take the designated amount of cod liver oil in the experimental period. He concluded, therefore, that while the administration of cod liver oil to the lactating mother is not a satisfactory method of preventing rickets in the young, a certain amount of antirachitic substance passes into the milk, raising the calcium-phosphorus quotient in the infant and lessening roentgenographic evidences of the disease.

A critical and practical analysis of this work leaves one with the impression that, as this method is of only slight value in antirachitic protection, and since the ingestion of cod liver oil by adults is often distasteful (as was pointed out in several of these articles), it is safer and more satisfactory in every way to feed the cod liver oil directly to the infant.

The effort to increase the antirachitic factor in human milk has not been limited to dietary regulation and ingestion of cod liver oil or other vitamin D concentrates. Hess [23] irradiated a lactating woman every other day for a month by exposure to ultraviolet rays. The vitamin D content of her milk seemed definitely increased. Previously, 25 cc. of her milk fed to rachitic rats had failed to cause any improve-

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21. Greenbaum, J. V.; Selkirk, T. K.; Otis, F. A., and Mitchell, A. G.: Effects of Diet During Pregnancy on Development of Rickets in the Offspring, J. A. M. A. **87**:1973 (Dec. 11) 1926.

22. Weech, A. A.: Influence of the Administration of Cod Liver Oil to the Mother on the Development of Rickets in the Infant, Bull. Johns Hopkins Hosp. **40**:244 (April) 1927.

23. Hess, A. F.; Weinstock, M., and Sherman, E.: Antirachitic Properties Developed in Human Milk by Irradiating the Mother, J. A. M. A. **88**:24 (Jan. 1) 1927.

ment. Following irradiation the same quantity caused definite improvement and raised the phosphorus content of the blood of the animals from an average of 1.98 mg. to 5.61 mg. per hundred cubic centimeters (the phosphorus content of the milk itself was not found to be increased). Hess pointed out that there was an increase in the nonsaponifiable fraction of the milk. He felt that the irradiation was of value not only to the baby, in that it increased the antirachitic content of the milk the infant was receiving, but also to the mother in preserving a correct mineral balance.

In approaching the actual problem of feeding the new-born one is met with the question as to when first to put the baby to the breast. Customarily this is done as soon as possible, usually, within twelve hours. Opposed to this is a highly interesting report by Oeljen and Scherer,[24] who compared two groups of infants, each group consisting of 200 babies. The first group was put to the breast within twelve to twenty-four hours following delivery, or an average of about fourteen hours; the second group had its first breast feeding in from twenty-four to thirty-six hours, or an average of about twenty-seven hours. Only 22 per cent of the first group went home at or above birth weight, while 34.5 per cent of the second group had attained or surpassed the birth weight on discharge. The average initial loss in the first group was 267 Gm., or 7.7 per cent of the birth weight; the average initial loss in the second group was 221 Gm. or 6.4 per cent of the birth weight. Greenwald and Pennell's work [25] offers some explanatory aid. They found no rise in the blood sugar of infants following breast feeding during the first forty-eight hours of life, probably owing to an insufficient secretion of breast milk. After the second day there was a definite rise in blood sugar, with a maximum usually in a half hour following feeding.

The problem of complementary feeding during the first few days of life is always troublesome. To complement or not to complement is the question. The feeling has been, possibly, that early complementary feeding tends to decrease the stimulation of the breast and consequently may cause a failure of breast feeding. The several reports concerning complementary feeding published during the past decade have been distinctly in its favor. Toothaker [26] compared a series of new-born infants who had received routine complementary feedings during the first few days of life with a similar series of new-born infants who

24. Oeljen, S. C. G., and Scherer, C. A.: First Application of New-Born Infant to Breast, Arch. Pediat. **50**:11 (Jan.) 1933.

25. Greenwald, H. M., and Pennell, S.: The Carbohydrate Metabolism of the Normal New-Born Infant, Am. J. Dis. Child **39**:493 (March) 1930.

26. Toothaker, J. E.: Routine Complementary Feeding of New-Born, Northwest Med. **29**:158 (April) 1930.

were solely breast fed. Eighty-two per cent of the first group had regained their birth weight on leaving the hospital (in an average of seven and seven-tenths days), while only 68 per cent of the second group had similarly attained their original weight on discharge (in an average of nine and eight-tenths days). Furthermore, inanition fever developed in only 2 per cent of the former, as compared with 11 per cent of the latter group. Dwyer and Neff [27] presented statistics even more favorable to complementary feeding. Only 31 per cent of solely breast-fed new-born infants regained their birth weight by the tenth day of life, while 68 per cent of those who received, in addition, complementary feedings—which were usually withdrawn after the third or fourth day—regained their original weight in the same period of time. Happ [28] believed that certain babies such as twins, premature infants, those under 6 pounds (2,721 Gm.) in weight and babies with sick mothers are definitely in need of complementary feeding. His complementary formula consisted of skimmed milk containing 10 per cent additional carbohydrate, for he felt that even young infants will tolerate relatively high percentages of carbohydrate if the fat content is kept low. In this respect the work of Griswold [29] on gastric digestion in new-born infants is interesting. He found that within a few hours following birth the stomach contains gastric juices with free hydrochloric acid at an average hydrogen ion concentration represented by $p_H$ 2.6. In five and ten days, removal of test meals at the height of digestion showed an average hydrogen ion concentration of $p_H$ 2.5, as compared with $p_H$ 3.9 to 4.6 in older infants. He concluded, therefore, that new-born and very young infants have an increased capacity for the secretion of hydrochloric acid and gastric digestion, and that more concentrated formulas than are now customary would be safe for them.

Kugelmass [30] devised an isotonic, isomolar solution consisting of 6 per cent neutral gelatin, 3 per cent dextrose and 0.5 per cent sodium chloride, which was fed to new-born infants every two hours. He showed that this solution corrected the hypoglycemia, decreased the blood volume and increased the viscosity of the serum which has been found in the blood of new-born infants, since gelatin is hydrating and easily assimilable, dextrose raises the blood sugar and the sodium chloride favors the retention of ingested water. By this means he

27. Dwyer, H. L., and Neff, F. C.: Complemental Feeding in the New-Born Infant, J. A. M. A. **99**:463 (Aug. 6) 1932.

28. Happ, W. M.: The Artificial Feeding of the New-Born Infant, California & West. Med. **23**:988 (Aug.) 1925.

29. Griswold, C., and Shohl, A. T.: Gastric Digestion in New-Born Infants, Am. J. Dis. Child. **30**:541 (Oct.) 1925.

30. Kugelmass, I. N.: Preventing Loss of Weight in the New-Born, Proc. Soc. Exper. Biol. & Med. **30**:164 (Nov.) 1932.

succeeded in reducing the initial loss from 10 per cent to 1.7 per cent of the birth weight, and believed that the solution was more satisfactory than ordinary complementary feeding.

### ARTIFICIAL FEEDING

*A. Acidified Milk.*—The use of acidified milk in infant feeding is not by any means an innovation of the past decade. It has been known and used for many years in the form of naturally soured milk. However, the literature of the past ten years has been particularly replete with brilliant investigations on this subject. Certainly its popularity in this country following Marriott's intensive work has been only a matter of a few years. If undue space is given in this paper to the discussion of acid milks, it is only because a large part of the literature has been devoted to that subject. In this discussion the review will touch on: (1) the theory of acid-milk feeding; (2) various methods of acidifying milk and a comparison of their products; (3) "the curd vs. the buffer," and (4) the relation of acid-milk feeding to mineral metabolism.

(*a*) The dependence of the action of enzymes on specific optimal conditions—in particular, the hydrogen ion concentration—is well known. The function of the gastric acid is to produce a concentration of hydrogen ions in the ingested food which will be optimal for the action of the gastric ferments or enzymes. The three chief gastric enzymes are rennet, lipase and pepsin. The ranges of optimal hydrogen ion concentration are different for all three. For rennet the range is $p_H \pm 5$, for lipase, from $p_H$ 5 to 4 and for pepsin, beginning at $p_H$ 4 but reaching optimal conditions at $p_H$ 2.8 to 2. For the human infant, milk, either human or bovine, is the chief food. Clark[31] was one of the first to show that cow's milk contains more buffers (which may be defined as substances tending to lessen changes in the hydrogen ion concentration on the addition of acids or alkalis) than human milk, and that considerable amounts of acid may be added to cow's milk without effecting a great change in the hydrogen ion concentration. These excess buffers are represented by the higher phosphate and caseinate content of cow's milk. While the addition of 30 cc. of decinormal hydrochloric acid to 100 cc. of breast milk changes the hydrogen ion concentration decidedly, from $p_H$ 6.9 to 3.4, the addition of the same amount of acid to a similar volume of cow's milk effects only a comparatively slight change in the hydrogen ion concentration, from $p_H$ 6.6 to 5.85. More than three times as much acid would be required to effect as great a change in the hydrogen ion concentration of cow's milk as of breast milk. In other words, an infant

31. Clark, W. W.: The Reaction of Cow's Milk Modified for Infant Feeding, J. M. Research **31**:431, 1914.

fed cow's milk must either secrete far more gastric acid than an infant receiving breast milk, or else not attain, in its stomach, the optimal conditions for digestion. Marriott [32] attacked the problem, not only theoretically but practically and experimentally, by determining the hydrogen ion concentration of the stomach in both normal and abnormal infants at the height of digestion following feedings of breast milk and of cow's milk. He summarized the results of his observations of the $p_H$ of gastric contents of normal infants and of those suffering from infection or nutritional disturbances as shown in table 3.

TABLE 3.—*$p_H$ of Gastric Contents*

| | Breast Milk | Cow's Milk | Acid Milk |
|---|---|---|---|
| Normal infants | 3.75 | 5.1 | 3.71 |
| Abnormal infants | 4.74 | 5.35 | 4.1 |

Obviously, as peptic digestion begins at $p_H$ 4, no peptic digestion is possible in abnormal infants ingesting breast milk or cow's milk, and even normal infants receiving cow's milk have but poor conditions for gastric digestion. (It should be added, however, that in a group of older infants accustomed to cow's milk, the gastric $p_H$ at the height of digestion, was 4.75, and Marriott felt that infants may gradually adapt the gastric secretion to this type of food so that a greater amount of hydrochloric acid is secreted.) The figures of most investigators have agreed fairly closely with those of Marriott and Davidson, although Wills and Paterson [33] reported somewhat lower findings. Their normal breast-fed infants showed an average gastric hydrogen ion concentration of $p_H$ 2.7; and the artificially fed, $p_H$ 3.5. However, they were in active agreement that slightly underweight infants have a lower acidity than normal babies, and sick infants show definitely lowered values of acidity. They concluded that in normal infants fed either breast milk or cow's milk the gastric acidity is such as to allow active peptic digestion, but that malnourished and sick infants may show a definite need for debuffering of the milk they ingest. Banu [34] showed that more combined acid and less free hydrochloric acid are present in the stomach of infants digesting cow's milk because of its greater buffer content, and that obviously cow's milk calls forth more work on the part of the

32. Marriott, W. M., and Davidson, L. T.: The Acidity of the Gastric Contents of Infants, Am. J. Dis. Child. **26**:542 (Dec.) 1923.

33. Wills, L., and Paterson, D.: A Study of Gastric Acidity in Infants, Arch. Dis. Childhood **1**:232 (Aug.) 1926.

34. Banu; Negresco, and Heresco: Le chimisme gastrique des nouveau-nés allaités au sein et de ceux nourris artificiellement, Compt. rend. Soc. de biol. **90**:1159 (May 9) 1924.

stomach. Müller [35] demonstrated a lowering of the blood chlorides in certain infants following feedings of cow's milk owing to the withdrawal of chlorides by the gastric glands in the excessive formation of hydrochloric acid.

Briefly, then, the buffer value of cow's milk is definitely higher than that of breast milk; far more gastric acid is needed, consequently, to achieve optimal hydrogen ion concentration when cow's milk is fed to infants; abnormal (malnourished and sick) infants and possibly even normal infants receiving cow's milk fail to achieve proper peptic digestion because they cannot secrete sufficient gastric acid both to debuffer the milk and to maintain an optimal hydrogen ion concentration.

As there is no practical method of increasing the gastric secretion of infants—even if that were advisable—one must have recourse to some method of debuffering the milk before feeding it. Dilution is one method by which this can be accomplished. If milk is diluted to one fourth its original concentration the buffer value will almost approximate that of breast milk. Obviously, however, this offers far too dilute a formula for most infants. The other, more practical method, is to debuffer the milk by the previous addition of some acid before ingestion by the baby, so that when the cow's milk reaches the infant's stomach, instead of being at $p_H$ 6.6, it is already at or near $p_H$ 4.

(*b*) Four acids have been proposed in this country for the artificial acidification and debuffering of cow's milk: lactic, hydrochloric, acetic (vinegar) and citric (lemon juice). Marriott, of course, has been the champion of lactic acid milk. His work is too well known to permit of anything but a few words of repetition here. Suffice it to say that the original method [36] consisted of adding 1 drachm (3.8 Gm.) of lactic acid, drop by drop, to 1 pint (473.1 cc.) of previously boiled, chilled milk. The later formula [37] was made with evaporated milk. A pint of water containing 1 drachm of lactic acid and 3 ounces (88.7 cc.) of corn syrup are added to a pint of evaporated milk, making a quart (946.3 cc.) of formula. Lactic acid milk may now also be prepared from dried lactic acid milk powder, which Hess [38] showed to be satisfactory.

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35. Müller, F.: Action of Artificially Soured Milk on Digestion and Metabolism of Infants; Influence on Gastric Digestion, Ztschr. f. Kinderh. **38**:704, 1924.

36. Marriott, W. M., and Davidson, L. T.: Acidified Whole Milk as a Routine Infant Food, J. A. M. A. **81**:2007 (Dec. 15) 1923.

37. Marriott, W. M.: Preparation of Lactic Acid for Infant Feeding, J. A. M. A. **89**:862 (Sept. 10) 1927.

38. Hess, J. H.; Chamberlain, I. M., and Robins, L. S.: Dried Lactic Acid Milks as a Long-Continued Diet for Infants, J. A. M. A. **98**:1250 (April 9) 1932.

Faber [39] advocated hydrochloric acid for the acidification of milk. His formula calls for 25 cc. of decinormal hydrochloric acid to 100 cc. of milk, the complete formula being: 960 cc. of milk, 240 cc. of decinormal hydrochloric acid and 60 cc. of corn syrup, having a hydrogen ion concentration represented by $p_H$ 6.

Dunham used vinegar [40] for acidification in the ratio of 1 ounce (29.5 cc.) of vinegar to 15 ounces (443.6 cc.) of milk, or a teaspoonful to 3 ounces of milk, producing a $p_H$ of 4.2. Later, acetic acid [41] itself—1 drachm to a pint of milk—was substituted. From 1 to 1½ ounces of additional carbohydrate were supplied to a pint of milk.

Hess [42] directed his efforts toward a method that would supply antiscorbutic factors as well as acidify the milk. At first tomato juice and then orange juice were tried, but while well taken, these substances were not sufficiently acid to effect much change in the hydrogen ion concentration of the milk unless comparatively enormous quantities were used. He finally decided in favor of lemon juice which contains from 6 to 7 per cent citric acid. His formula consisted of: milk, 24 ounces; water, 12 ounces; sucrose, ½ ounce, and lemon juice, 21 cc. Its hydrogen ion concentration is represented by $p_H$ 5.3. For older infants whole milk may be used. Then Hess recommended the further addition of one egg yolk to the formula, thus supplying added antirachitic factors (and incidentally markedly increasing the iron intake of the infant).

Some further modifications of lemon juice, or citric acid, milk have been advocated. Gonce and Templeton [43] used dehydrated citric acid, adding 4 Gm. of the acid to a quart of boiled milk cooled to room temperature. Lynch [44] used reliquefied powdered citric acid milk. The powder, originally prepared by adding 4 Gm. of acid to a quart of milk ($p_H$ 4.5), caused some vomiting and gastric upset. This was corrected by reducing the amount of citric acid to 2 Gm.

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39. Faber, H. K.: Hydrochloric Acid Milk in Infant Feeding, Am. J. Dis. Child. **26**:401 (Nov.) 1923.

40. Dunham, B. S.: Acidification of Milk with Vinegar (Acetic Acid) in Infant Feeding, Am. J. Dis. Child. **29**:200 (Feb.) 1925.

41. Dunham, B. S.: Acetic Acid Milk from Acetic Acid of U. S. Pharmacopeia, Am. J. Dis. Child. **32**:384 (Sept.) 1926.

42. Hess, A. F., and Matzner, M. J.: Value of Milk Acidified with Lemon Juice: Its Combination with Egg-Yolk to Add the Antirachitic Factor, J. A. M. A. **82**:1604 (May 17) 1924.

43. Gonce, J. C., and Templeton, H. L.: Citric Acid Milk in Infant Feeding, Am. J. Dis. Child. **39**:265 (Feb.) 1930.

44. Lynch, H. D.: Powdered Citric Acid Milk: Comparison of Clinical Results with Those of Powdered Whole Milk, Arch. Pediat. **49**:763 (Nov.) 1932.

Reiss [45] used two formulas which included lemon juice and evaporated milk. The routine formula consisted of: evaporated milk, 14 ounces; water, 16 ounces; corn syrup, 2 ounces, and lemon juice, 5 drachms, with a $p_H$ of 5.35. A slightly weaker formula was given to younger and new-born infants, consisting of: evaporated milk, 14 ounces; water, 20 ounces; corn syrup, 1½ ounces, and lemon juice, 5 drachms, with a $p_H$ of 5.6.

What are the respective advantages and disadvantages of these acid milks; what determined their choice? Marriott used lactic acid because it is the acid of naturally soured milk. Hydrochloric acid was advocated by Faber because it is the physiologic acid of gastric secretion. Dunham, seeking an acid that would be inexpensive and a common household commodity, decided on ordinary vinegar. Hess, as has been seen, wishing to include antiscorbutic elements in the process of acidification, finally chose lemon juice. Possibly the most controversial and important point is the question of degree of acidification or debuffering. Marriott's lactic acid milk at $p_H$ 4 is the most highly acidified, with the vinegar milk at $p_H$ 4.2 not far behind. Hess' lemon juice milk at $p_H$ 5.3 holds an intermediary position, but Faber's hydrochloric acid milk at $p_H$ 6 is obviously only partially debuffered. Faber [46] ardently upheld the partially debuffered milk as the more physiologic rationale. He believes that the ingestion of a food already at or near the optimal hydrogen ion concentration removes the need of, and hence the stimulus to, the normal secretion of hydrochloric acid by the stomach. He expressed the opinion that for routine feeding in cases of mild digestive disorder, the use of a milk containing approximately the same amount of buffer substances as breast milk and hence requiring the normal secretory effort for which the stomach is adapted would seem to be the physiologic method par excellence. In conditions of marked deficiency of the gastric secretory ability, as in marasmus, complete debuffering is more logical. Another advantage of partial debuffering is that it permits all the gastric ferments to exert their maximal effect in order, whereas if food, on ingestion, is already at $p_H$ 4, optimal conditions for rennet and lipase action are already passed over. Faber, in fact, did not advocate acidified milk for routine feeding of infants, as did Marriott, but restricted its use to specific indications, such as marasmus, diarrheal conditions and tetany. Many persons, as a survey by Levy [47] showed, do not adhere strictly to Marriott's formula, and are only par-

45. Reiss, O.: Lemon Juice Evaporated Milk in Infant Feeding, Arch. Pediat. **49**:170 (March) 1932.

46. Faber, H. K.: Acidified Milks with Special Considerations of the Indications and Limitations to Their Use, Am. J. Dis. Child. **31**:395 (March) 1926.

47. Levy, D. J.: Variable Factors in Lactic Acid Milk Feeding, J. Michigan M. Soc. **26**:150 (March) 1927.

tially debuffering milk by using lactic acid varying in amount from 15 to 240 drops. Levy, too, felt that for ordinary use a partially debuffered milk is best.

While Faber's tenet is strong it would seem from other reports that his choice of acid was unfortunate. Levine [48] stated that hydrochloric acid milk is sour to the taste and may often be refused; the addition of the acid may cause a heavy precipitate of flocculent casein; and as hydrochloric acid is an inorganic acid, it is not oxidized, as are the others, but effects a reduction in the alkaline reserve of the body.

Of distinct importance, naturally, is the further fate and action of these acids in the body. In a comparison of lactic acid and hydrochloric acid milks, it was found that the latter exerted a definite diuretic action, resulting in an excretion of from 20 to 30 per cent more urine. Furthermore, the total acidity of the urine, which is the measure of the acid effect produced by any ingested acid, is markedly increased by hydrochloric acid milk, but is practically unaffected by feedings of lactic acid milk, presumably because lactic acid is an organic acid and is completely oxidized before reaching the kidneys. Darrow [49] found no significant changes in the bicarbonate, lactate or hydrogen ion concentration of the blood following feedings of ordinary or lactic acid milk. Brahdy,[50] corroboratively, showed that the lactate concentration of the blood for two or three hours following the ingestion of lactic acid milk was essentially the same as after feedings of sweet milk. Pursuing further the question of the effect of acid milks on the kidneys, Greene [51] found that hydrochloric acid frequently produced casts and occasional red blood cells in the urine, especially in younger infants. As little as 45 cc. of decinormal acid to a quart of milk would at times produce these urinary changes. Von Bernuth and Duken [52] reported that 14 of 23 infants receiving hydrochloric acid milk had casts in the urine.

In favor of acetic acid is the fact that the acetates which are formed are more rapidly absorbed than the lactates or citrates, thus exerting less laxative effect. The curd formed is fine, smooth and flocculent. Acetates seem to be devoid of any specific action, as they are completely oxidized to carbon dioxide and water. The antiscorbutic value of citric

48. Levine, V. E.: Modern Aspects of Nutrition, Nebraska M. J. **11**:100 (March) 1926.

49. Darrow, D. C.: Carbon Dioxide Absorption Curve of Infants Fed Lactic Acid, Hydrochloric Acid and Boiled Cow's Milk, Am. J. Dis. Child **40**:1016 (Nov.) 1930.

50. Brahdy, M. B.: Lactate Metabolism in Infants: Its Relation to Lactic Acid Milk, Am. J. Dis. Child. **33**:451 (March) 1927.

51. Greene, D.: Effect on the Urine of Addition of Acids and Alkalies to the Diet of Infants, Am. J. Dis. Child. **35**:38 (Jan.) 1928.

52. von Bernuth, F., and Duken, J.: Klinische Beobachtungen über Stuhlbeschaffenheit und Zylindrurie bei Salzaüremilch, Arch. f. Kinderh. **80**:21, 1926.

acid milk is unquestionably a large factor in its favor. The urine is not affected. As Hess pointed out, citric acid is a normal constituent of cow's milk, the percentage content increasing when the fodder is changed from stall feeding to grazing. The addition of egg yolk contributes to its antirachitic value, making it practically a complete food for infants. Levine [48] also advocated the addition, in goitrous districts, of small amounts of iodine—0.125 mg. to a quart of milk.

Clinically, lactic acid milk has unquestionably had the widest use. What do purely clinical reports tell one of the comparative merits of these milks? Actual comparative series have been few. Green and his associates,[53] in a series of tests of feeding, found the lemon juice milk a little more readily taken at the onset than lactic acid milk. However, infants suffering from various digestive disturbances of an acute nature seemed more rapidly brought to normal by lactic acid milk than by the other acid milks. Hydrochloric acid milk was fed to a small group of infants who did well, but there was a tendency to excoriation of the buttocks. Gonce and Templeton [43] believed that their citric acid milk was retained better than lactic acid milk. The curds are soft and small, mostly less than 1 mm. in diameter, whereas the curds of lactic acid milk tended to fuse into larger masses.

Elsewhere the use of artificially acidified milks has not been as extensive as in this country. Lactic acid milk has been used almost exclusively, although in Germany citric acid milk [54] has also been used. The naturally soured rather than the artificially prepared lactic acid milk has been more popular in Germany, especially recently, in the form of powdered buttermilk,[55] the advantages of which are ease of handling and use and constancy of the product. Marfan [56] concluded from his

53. Green, H.; Withington, P. R., and Friedman, L. S.: Acid Milk in Infant Feeding, Boston M. & S. J. **192**:588 (March 26) 1925.

54. Meyer, S.: Zitronensäurevollmilch in der Diätetik de Ruhr im Kindesalter, Arch. f. Kinderh. **82**:241 (Nov. 7) 1927. Weissenberg, H.: Die Behandlung der Säuglingsatrophie mit der Zitrotibinmilch, ibid. **90**:288 (June 27) 1930. Kochmann, R., and Steinbach, M.: Klinische Erfahrungen mit Milchsäurevollmilch und Zitronensaftvollmilch, Jahrb. f. Kinderh. **118**:36 (Nov.) 1927. Weissenberg, H.: Weitere Erfahrungen über Rachitis-Prophylaxe mit der Zitrotibinmilch, Arch. f. Kinderh. **97**:93 (July 22) 1932. Niermann, M., and Winter, M.: Stoffwechseluntersuchungen bei Ernährung mit sauren Milchmischungen, ibid. **89**:290 (Feb. 28) 1930. Behrens, R.: Erfahrungen mit der Zitronensäurevollmilch Weissenbergs, im besondern bezüglich ihrer prophylactischen Wirkung auf Rachitis, ibid. **86**:90 (Jan. 25) 1929.

55. Engel, S.: Zur Praxis der Säuglingsernährung mit Buttermilch, Deutsche med. Wchnschr. **56**:433 (March 14) 1930. Gierthmühlen, F.: Erfahrungen mit Trockenbuttermilch, ibid. **54**:1678 (Oct. 5) 1928. Langstein, L.: Buttermilch in Pulverform, ibid. **53**:957 (June 3) 1927.

56. Marfan, A. B., and Chevalley, M.: Essai de substitution du lait acidifié au babeurre dans l'alimentation des nouveau-nés débiles et hypothrepsiques, Bull. Soc. de pédiat. de Paris **26**:239 (May 15) 1929.

clinical experience that buttermilk or a combination of buttermilk and breast milk is far superior to lactic acid milk. Craig and Tobey [57] advocated cultured lactic acid milk because they felt that the distribution of acid is more uniform and homogeneous, as each organism is a center for the dissemination of acid which is quickly and evenly distributed, with no tendency for curds to fuse into masses. Hess [58] concluded that cultured lactic acid milk stands by itself as regards digestibility, as, following digestion in vitro, the increase in soluble nitrogen (the measure of peptic digestion) is 100 per cent more than in raw certified milk. Lemon juice milk shows about 25 per cent increased digestion over raw certified milk. Müller, however, felt, from similar experiments in digestion in vitro, that the protein of the chemically acidified milk was digested better than the bacterially soured product. In England lactic acid milk has been almost the sole acidified milk used. Most reports have been favorable, but there has been a feeling [59] that in the treatment of certain groups of intestinal upsets such as gastro-enteritis and ileocolitis the Finkelstein albumin milk is superior.

(*c*) As the question of "the curd vs. the buffer" is approached one sees that certain investigators have directed a counter-attack on the importance of the debuffering action of acids. In a fairly recent publication Brennemann [60] discussed the relative importance of the curd and the buffer in infant feeding. He believed that in the acidification of milk two radical changes take place: reduction of the buffer content and attenuation of the curd. In an attempt to decide which effect was more important he conducted a series of feeding experiments consisting of alternate periods of feeding with evaporated milk and with acidified evaporated milk. In the former, only the curd is affected, while in the latter, both the curd and the buffer values are altered. The consensus of all observers was that the various periods were indistinguishable. There was no difference in gain in weight or in other clinical criteria. Consequently, Brennemann concluded that the debuffering action of the acid plays but a small rôle, and the attenuation of the curd is of major importance.

Lynch,[44] in a comparable series of routine feeding, offered reliquefied dried milk and reliquefied citric acid dried milk to two groups of infants. The clinical course and average gain in weight were much the same for

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57. Craig, J. D., and Tobey, J. A.: The Advantages of Cultured Lactic Acid Milk, Arch. Pediat. **49**:534 (Aug.) 1932.

58. Hess, J. H.; Koch, E. M., and Sennewald, Z. C.: Peptic Digestion of Cow's Milk: The Effect of Various Modifications Used in Infant Feeding, J. A. M. A. **87**:1360 (Oct. 23) 1926.

59. Thatcher, L.: Acidified Milk for Infant Feeding, Edinburgh M. J. **37**:37 (Jan.) 1930.

60. Brennemann, J.: The Curd and the Buffer in Infant Feeding, J. A. M. A. **92**:364 (Feb. 2) 1929.

the two groups. The group receiving citric acid milk made an average gain of 21½ ounces, and the other group, 22 ounces a month. It seemed logical to conclude that when cow's milk is altered by physical means such as drying (again attenuation of the curd) there is less need for acidification to render further changes in digestibility (one must keep in mind, however, that acidified milk was being used here as a routine and not as a therapeutic food. Would the same premise be applicable to the feeding of malnourished and sick infants?) Lynch pointed out that Marriott originally used 8 cc. of lactic acid in the preparation of a quart of ordinary acid milk, but only 4 cc. of acid for a quart of formula prepared from evaporated milk.

Hess [58] complicated this further by demonstrating by means of experiments in digestion in vitro that aside from the physical character of the curd there are several other factors which influence the peptic digestion of the proteins of milk, and that the previous denaturization of the protein by heat, which takes place in the evaporation process, plays an important rôle. Lautz,[61] however, in a similar experiment, concluded that neither the addition of acid nor heating had much effect on peptic digestion.

Holm and Webb,[62] too, believed that the nature of the curd formed was of greater importance than the buffer capacity, pointing out that goat's milk has a relatively high buffer capacity and yet is well tolerated in many difficult cases of feeding because of its finely divided curd (due, in large measure, to its relatively high percentage of albumin). Dennett and Craig's [63] comparison of several groups of infants is also of interest. The group receiving evaporated milk made an average weekly gain of 5.7 ounces, whereas the group receiving ordinary milk gained only 4.9 ounces a week. They pointed out that the curd tension of restored evaporated milk (only 3 Gm.) is far lower than that of ordinary milk.

The question of the curd is further elaborated by Hill,[64] who showed that cows differ greatly in the type of curd formed by their milk, the curd tension as recorded by the Hill test varying from 10 to 250 Gm. Most prepared milks have a curd tension of from 60 to 90 Gm. He felt that milk with a curd tension of less than 20 Gm. was particularly desirable and could be used without dilution or modification. Elias,[65] however, in a clinical experiment, found no advantages in the use of

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61. Lautz, A.: Effect on Peptic Digestion in Vitro of Artificial Feeding as Used for American Infants, Am. J. Dis. Child. **45**:985 (May) 1933.

62. Holm, G. E., and Webb, B. H.: Buffer Capacities of Various Milks and Proprietary Infant Foods, Am. J. Dis. Child. **40**:260 (Aug.) 1930.

63. Dennett, R. H., and Craig, J. D.: A Comparative Study of Infant Foods, M. J. & Rec. **136**:134 (Aug. 17) 1932.

64. Hill, R. L.: Soft-Curd Milk: Nature's Food for Infants, Arch. Pediat. **48**:417 (July) 1931.

65. Elias, H. L.: Soft-Curd Milk, Am. J. Dis. Child. **44**:296 (Aug.) 1932.

milk with a soft curd. Infants fed this type of milk showed the same gains in weight as did a control group. There seemed to be no advantage in digestibility or in preventing or treating vomiting and gastrointestinal upsets.

Blackfan and Johnston [66] questioned whether the increased gain in weight achieved in malnourished infants by the use of acidified milk was due simply to the acidification or to the fact that mixtures of whole milk with high percentages of additional carbohydrate, i. e., concentrated feedings high in caloric value, were being offered. Their two groups of infants, one of which was fed acidified milk and the other, formulas of sweet milk of equal caloric value, showed practically identical gains in weight, averaging 0.59 ounce daily for the former and 0.61 ounce daily for the latter group. Dwyer's [67] experience, in a similar experiment, was the same.

What can one conclude from this mass of fact, fancy and foible? If one is to judge by the work of R. Hess,[68] the whole question has had an exaggerated amount of attention, as he concluded that in infants gastric digestion is, after all, of but slight importance. It is obvious that the last word has yet to be written. It is equally obvious that definite conclusions are at best to be drawn with hesitancy and difficulty. It seems that the battle of the curd vs. the buffer is still being waged. Routine feeding of acid milk would seem to be superfluous, as infants seem to get along equally well on sweet milk. However, in marasmus and other conditions in which it has been shown that the infant's gastric secretion is at fault it seems only logical to use debuffered milk—one approaching a hydrogen ion concentration of $p_H$ 4 rather than a partially debuffered food. It seems equally logical to presume that in these cases, reemphasizing that the formulas are not for routine feeding but for infants whose gastric secretory ability is below par, the acidification per se plays the major rôle through its debuffering power rather than through its attenuation of the curd.

What are some of the other advantages that have been claimed for acidified milk? Briefly, they are:

1. Its antiseptic action. It has been shown that a hydrogen ion concentration of $p_H$ 5 inhibits the growth of the colon-dysentery-typhoid group of organisms, while one of $p_H$ 4 results in the death of these bacteria. Judging from Schiff and Gottstein's [69] experiment, this anti-

66. Blackfan, K. D., and Johnston, J.: Notes on Infant Feeding, Boston M. & S. J. **196**:395 (March 10) 1927.

67. Dwyer, H. L.: Acidified Milk in Infant Feeding, J. Kansas M. Soc. **26**: 140 (May) 1926.

68. Hess, R.: Ueber die Zunahme gelösten Stickstoffs im Säuglingsmagen, Monatschr. f. Kinderh. **36**:208, 1927.

69. Schiff, E., and Gottstein, W.: Acidity in Duodenum Contents, Jahrb. f. Kinderh. **107**:99 (Sept.) 1924.

septic action must presumably be limited to the stomach. In an attempt to discover whether the beneficial effects of buttermilk in nutritional disturbances associated with diarrhea were due to the inhibition of the growth of Bacillus coli by the acidity of the food, they fed various types of milks to dogs with Eck fistulas. When neutral food was given, the duodenal contents were acid; after the ingestion of acidified food the duodenal contents were alkaline, and in all cases the contents of the lower third of the jejunum were alkaline in reaction. Thus, they concluded that the therapeutic value of buttermilk did not depend on its power to inhibit the growth of B. coli in the intestine.

2. The regulation of gastric motility. It has been found that a larger amount of food remains in the stomach at the end of two hours in patients with gastric hypo-acidity than in normal persons. Thus, in malnourished and sick infants, feedings of acid milk are also of aid in emptying the stomach within a reasonable length of time.

3. Greater stimulation to the flow of pancreatic juices, bile and intestinal secretions by the acid chyme.

4. Increased mineral retention. The review of this subject will be incorporated in the discussion of mineral metabolism.

*B. Protein Milk.*—In this furore of acid milk feeding, further work on protein milk has been more or less neglected, in this country at least, during the past decade. In fact, its efficacy in certain diarrheal conditions has been attributed by Levine [70] to the very fact that it is essentially a product of soured milk with a hydrogen ion concentration of about $p_H$ 4.5, rather than to its elevated protein and low lactose and salt content. Since Meyer's report on the injurious effect of a mixture of human milk casein and cow's milk whey and the harmlessness of a similar mixture of human milk whey and cow's milk casein and Finkelstein's investigations on protein milk feeding, little further has been added to the knowledge of the theory and practice of protein milk therapy in infant feeding.

However, an interesting series of articles elaborating somewhat on the injurious effects of cow's milk whey has come from Finkelstein's clinic.[71] It was found, by following the course of blood sugar curves, that cow's milk whey caused a delay in the absorption of food elements by the intestine. These investigators showed that the absorption of sugar, as measured by the effect on blood sugar levels, was normal with

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70. Levine, V. E.: Acidified Milks in Infant Feeding, Arch. Pediat. **43**:631 (Oct.) 1926.

71. Finkelstein, H.: Whey as Factor in Infants' Diarrhea, Ztschr. f. Kinderh. **39**:1, 1915. Davidsohn, H.: Whey and Lactase, ibid. **40**:251 (Nov. 13) 1925. Keilmann, K.: Whey and Blood Sugar, ibid. **40**:259 (Nov. 13) 1925. Bernhard, E.: Whey and Lactosuria, ibid. **40**:275 (Nov. 13) 1925. Finkelstein, H.: Clinical Study, ibid. **40**:277 (Nov. 13) 1925.

human milk; whole cow's milk, however, caused the same delay in absorption that whey did. On dilution of whole milk with water in the proportion of 1:2 this delay disappeared. Protein milk, with only half the whey content of ordinary milk, showed less interference with absorption than whole whey. The conclusions were that delayed absorption by the intestinal tract was caused by some injury to the intestinal cells by the foreign (cow's) whey. Injury to the intestinal mucous membrane was further evidenced by the fact that the cells were made more permeable to lactose, as shown by the appearance of lactosuria. In brief, it was felt that cow's milk whey has an influence in the origin of digestive upsets in infants.

Aside from these reports there is little new and important. Somewhat beyond the realm of practical value are the several reports on the effect of feedings high in protein. Eckert [72] found that diets containing about 30 per cent protein (a certain amount of sugar being replaced by lactalbumin) affected digestion and caused loss of weight. The stools were often foul and mucous. Experiments in animals by Reader and Drummond [73] showed that when rats were fed diets high in protein (45 to 90 per cent) they maintained fairly good health, but failed to grow to adult size. Paul [74] showed that if a child receiving ordinary feedings of protein milk fails to gain weight and shows persistent indicanuria, it is advisable to change the proportions of protein and carbohydrate by increasing the latter. That protein milk may, however, be used for long periods with good results was demonstrated by Haas and his co-workers,[75] who recorded an average weekly gain of 4 ounces for infants of all ages on prolonged feedings of protein milk.

*C. Evaporated, Dried and Condensed Milk.*—The increasing popularity of evaporated milk in infant feeding has been an outstanding feature of the newer developments. What has concerned most of the workers on the subject has been, naturally, how this milk compares in digestibility, growth-promoting qualities and other nutritional factors with ordinary milk. Marriott [76] summarized some of the general characteristics of evaporated milk. It is made by evaporating about 60 per cent of the water of ordinary milk, after which it is sterilized in the

72. Eckert, M.: Effect of Concentrated Albumin in Infant Feeding, Ztschr. f. Kinderh. **37**:1, 1924.

73. Reader, V. B., and Drummond, J. C.: Further Observations on Nutrition with Diets Rich in Protein, J. Physiol. **59**:472 (March) 1925.

74. Paul, S.: Indicanuria as Guide in Treatment with Albumin Milk, Monatschr. f. Kinderh. **27**:504 (Feb.) 1924.

75. Haas, S. V.; Barenberg, H. H., and Bloomberg, N. W.: Protein Milk as Infant Food, Arch. Pediat. **41**:751 (Nov.) 1924.

76. Marriott, W. M., and Schoenthal, L.: An Experimental Study of the Use of Unsweetened Evaporated Milk for the Preparation of Infant Feeding Formulas, Arch. Pediat. **46**:135 (March) 1929.

autoclave at a temperature of from 200 to 240 F. This heating gives rise to certain alterations in the chemical, physicochemical and physical properties. The casein undergoes alteration, so that when subsequently subjected to coagulation, the curd is much finer than in raw, pasteurized or quickly boiled milk. A large or tough curd cannot be formed from evaporated milk. Dennett and Craig reported the following relative curd tensions: certified milk, 42 Gm.; certified milk boiled two minutes, 30 Gm.; soft curd certified milk, 28 Gm.; soft curd certified milk boiled two minutes, 18 Gm., and restored evaporated milk, 3 Gm.

Not only is the digestion of the protein facilitated by the physical changes in the curd, but also by the denaturization of the protein which takes place during the heat process.[58] Wallen-Lawrence and Koch [77] showed by experiments with tryptic digestion in vitro that when raw milk is pasteurized, boiled or evaporated, it undergoes changes which make it more readily digested. The change is greater for boiled and evaporated than for pasteurized milk and seems to be a function of the temperature and the length of time of heating to which the milk is subjected. They showed further that the differences in tryptic digestibility were due to some heat-labile constituent of the whey, probably in the albumin portion, which they called the tryptic inhibitor, and assumed that it reacted monomolecularly with trypsin to inactivate it.

Nevens and Shaw,[78] however, while realizing the great amount of work that had been done demonstrating the "ease of digestibility" of the protein fraction of evaporated milk as compared with fresh milk, decided to investigate the relative "completeness of digestion" of the two. They found that in rats the protein of fresh milk was distinctly more completely digested than that of evaporated milk. They were at a loss to explain why the evaporated milk proved superior for growth.

The heating that evaporated milk undergoes does not alter the chemical character of the fat, but the globules are broken up into much finer particles. The carbohydrate fraction remains unaffected. In the heating process there may occur a slight precipitation of calcium phosphate. Rice [79] showed, however, that when evaporated milk is restored to its original volume, the electric conductivity is within the limits observed for ordinary milk (from 40 to $45 \times 10^{-4}$), indicating that the soluble mineral constituents have not been greatly altered. Furthermore, it has been shown that the precipitated phosphate is soluble in the digestive

77. Wallen-Lawrence, Z., and Koch, F. C.: The Relative Digestibility of Unsweetened Evaporated Milk, Boiled Milk and Raw Milk by Trypsin in Vitro, Am. J. Dis. Child. **39**:18 (Jan.) 1930.

78. Nevens, W. B., and Shaw, D. D.: The Effect of Dairy Manufacturing Processes upon the Nutritional Value of Milk: I. The Apparent Digestibility of Fresh Whole Milk and of Evaporated Milk, J. Nutrition **5**:485 (Sept.) 1932.

79. Rice, F. E.: The Use of Unsweetened Evaporated Milk in Infant Feeding, Arch. Pediat. **44**:758 (Dec.) 1928.

juices and consequently digestible and absorbable. A majority of the reports have indicated that the retention of calcium and phosphorus is as good from evaporated milk as from ordinary milk. Willard stated that calcium, phosphorus and nitrogen are retained by both adults and children equally well from evaporated as from ordinary pasteurized milk. Willard and Blunt,[80] in metabolic experiments on 4 children, using three day test periods, found higher phosphorus retention in all 4 and higher calcium and nitrogen retention in 3 when they were being fed evaporated milk, as compared with pasteurized milk. Kramer, Latzke and Shaw [81] offered one of the few objections. They used a group of children and adults as experimental subjects for a comparison of raw, pasteurized, evaporated and dried milks as sources of calcium and phosphorus. Five children showed their best retention of calcium when ingesting fresh milk. Four of 6 adults, however, showed better retention of calcium from evaporated than from fresh milk. Phosphorus retention followed the same trend.

Marriott also discussed the vitamin content of evaporated milk. He pointed out that the fat-soluble vitamins A and D are fairly stable and resistant to heat and do not deteriorate on standing, and that evaporated milk retains these two vitamins in practically their original concentration. Heating destroys to a considerable extent the variable amount of vitamin C in cow's milk, but as antiscorbutic adjuvants are offered as a routine in the modern dietary of the infant, this loss of vitamin C is not a great disadvantage. A good deal of controversy has existed regarding the amount of destruction of vitamin B in evaporated milk. Some of the earlier experiments are confusing because of the lack of differentiation between the two factors in vitamin B, $B_1$ and $B_2$ or F (the antineuritic factor) and G (the growth-promoting factor). Marriott stated that $B_1$ is thermolabile in alkaline solutions, but is not readily destroyed even when subjected to high temperatures in slightly acid mediums, and that it has not been possible to demonstrate any considerable diminution of vitamin $B_1$ content in evaporated milk as compared with fresh milk by direct experiments in feeding with young animals. He drew his conclusions from some earlier experiments of Dutcher. However, later work [82] seemed to indicate that there is some

80. Willard, A. C., and Blunt, K.: A Comparison of Evaporated with Pasteurized Milk as a Source of Calcium, Phosphorus and Nitrogen, J. Biol. Chem. **75**:251 (Oct.) 1927.

81. Kramer, M. H.; Latzke, E., and Shaw, M. M.: A Comparison of Raw, Pasteurized, Evaporated and Dried Milks as Sources of Calcium and Phosphorus for the Human Subject, J. Biol. Chem. **79**:283 (Sept.) 1928.

82. Hartwell, G. A.: A Comparison of Dried and Evaporated Milks by a Dietetic Method, Biochem. J. **19**:226, 1925. Daniels, A. L., and Brooks, L.: Further Evidences of Destruction of Vitamin B in Evaporated Milk, Proc. Soc. Exper. Biol. & Med. **25**:161 (Dec.) 1927.

destruction of the $B_1$ factor in the process of evaporation. Daniels [83] believes that the destruction of the antineuritic element is related to the time of exposure to high temperature and to the degree of aeration. All superheated milks except milk dried by the roller process showed considerable destruction of the $B_1$ element; open pasteurized milk showed some loss of the vitamin but much less than evaporated or spray-dried milk. Milk pasteurized by the closed method seemed unaffected. More recent work by Samuels and Koch [84] seems to indicate that about one fifth of the antineuritic factor in milk is lost in the process of evaporation. Of how much practical importance this moderate loss is in infant feeding is still questionable. $B_2$ has been shown to be remarkably heat stable. Thus, evaporated milk has the same nutritional value as fresh milk and has the further advantages of sterility, easy digestibility and uniformity of composition.

Clinical reports attesting to the efficacy of this milk in infant feeding are numerous. Marriott reported a series of new-born infants fed evaporated milk formulas exclusively. Ninety-two and eight-tenths per cent of the series regained their birth weight by the fourteenth day of life, while only 66 per cent of a group of controls fed at the breast or at the breast with complementary feeding had regained their birth weight in the same length of time. This can probably be explained by the somewhat greater average caloric intake by the former group, but it shows nevertheless that evaporated milk is readily utilized even by very young infants. A group of 9 premature infants fed exclusively evaporated milk formulas made an average daily gain of 0.92 ounce. Others, as has been previously demonstrated, have also had good results in the feeding of premature infants with evaporated milk alone or as an addition to breast milk.

Judging from Dennett and Craig's experience,[63] evaporated milk seems to be clinically superior to both ordinary cow's milk and breast milk in routine feeding. They fed four groups, of approximately 100 infants each, breast, cow's, evaporated and dried milk, respectively, for from nineteen to twenty-six weeks. The average weekly gain in ounces for each group was: breast milk, 4.95 Gm.; modified cow's milk, 4.93 Gm.; evaporated milk, 5.7 Gm., and dried milk, 5.7 Gm. Kositza [85] found that especially in the earlier months of life there were greater

83. Daniels, A. L.; Giddings, M. L., and Jordan, D.: The Effect of Heat on the Antineuritic Vitamin of Milk, J. Nutrition **1**:455 (May) 1929.

84. Samuels, L. T., and Koch, F. C.: The Relative Quantities of the Heat-Stable and Heat-Labile Fractions of Vitamin B in Raw and Evaporated Milk, J. Nutrition **5**:307 (May) 1932.

85. Kositza, L.: A Comparative Study of the Use of Unsweetened Evaporated Milk and Bottled Cow's Milk in Infant Feeding, J. Pediat. **1**:426 (Oct.) 1932.

gains made by infants fed evaporated milk than by those fed ordinary milk. Developmental phenomena such as the time of onset of teething and walking were the same for both groups.

Sauer [86] devised a simple stock formula of evaporated milk which may be used throughout early infancy, increasing the volume at feedings as the baby grows older. This formula consists of: evaporated milk, 6 ounces; water, 12 ounces, and lactose, 1 ounce. Evaporated milk has, of course, been widely used for acid milk formulas, and ease in the preparation of acidified milk has been enhanced by the use of evaporated rather than ordinary milk.

Dried milk has likewise been used a great deal in the past few years. The reports have been almost uniformly favorable. In Dennett and Craig's table, one can see that the babies receiving dried milk formulas made better gains than those receiving breast or cow's milk. Amick's [87] results after feeding 100 infants powdered milk formulas are similar; the babies showed an average weekly gain of 5¾ ounces during the first six months of life and 4¼ ounces during the second half of the first year. Even better results are seen in Ashton and Stringfield's [88] series of infants, which averaged a weekly gain of 6¾ ounces in the first six months and 4¾ ounces in the second six months of life. Of interest is the report by Supplee and his associates [89] which showed that milk dried by the roller process contained from two to five times as much iron as ordinary milk, owing to the dissolution of iron from the metallic cylinders. The characteristic milk anemia that develops in animals fed ordinary milk did not develop in rats to which this roller-dried milk was fed, and the iron content of the bodies of the animals fed dried milk was much higher than that of the animals used as controls.

From France in particular reports [90] have come concerning fever occurring occasionally in infants receiving dried milk formulas. While the infants gained in weight and were otherwise without symptoms, an unexplainable fever would occasionally develop. This slight increase

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86. Sauer, L.: A Simple, Inexpensive Stock Formula for Young Infants, J. Pediat. **1**:194 (Aug.) 1932.

87. Amick, A. E.: A Clinical Study of Powdered Whole Milk in Infant Feeding, West Virginia M. J. **28**:196 (May) 1932.

88. Ashton, L. O.; Stringfield, O. L., and Martin, C. W.: A Study of the Routine Use of Powdered Whole Milk in Infant Feeding, Arch. Pediat. **46**:75 (Feb.) 1929.

89. Supplee, G. C.; Dow, O. D.; Flanigan, G. C., and Kahlenberg, O .J.: A Comparative Study of Liquid and Dry Milk as Anemia-Producing Diets, J. Nutrition **2**:451 (May) 1930.

90. (*a*) Dorlencourt, M. H.: Faits cliniques et expérimentaux concernant la fièvre de lait sec, Bull. Soc. de pédiat. de Paris **27**:421 (Oct. 15) 1929. (*b*) Marfan, A. B.: Le lait concentré et le lait sec, Nourrisson **18**:1 (Jan.) 1930. (*c*) Lereboullet, M. P.: La fièvre de lait sec, Progrès méd., July 25, 1931, p. 1333.

in temperature never appeared immediately after giving the baby a dried milk formula. There was usually a latent period varying from several days to months; but if the infant had once previously been fed powdered milk, the fever would develop almost immediately on resumption of the same powdered milk, but not on the use of any other. The theory that there might be some toxic substance formed in the process of manufacture could not be supported experimentally. Dorlencourt [90a] thought it was due to a sensitization to some substance formed in the process of drying. Lereboullet,[90c] however, believed there was no anaphylactic reaction, but that the fever was due simply to insufficient water in the infant's diet. From the description given, Dorlencourt's theory seems more reasonable.

Acidified dried milks have been fairly popular and successful. Blatt and Nichamin [91] reported that powdered lactic acid milk proved to be the most successful in a series of difficult feeding cases.

Condensed milk, which has been looked on by pediatricians, although not by the laity, more or less as a poor relation in infant feeding, may, according to some recent reports, be more sinned against than sinning. DeSanctis and Craig [92] gave a distinctly favorable report on condensed milk. In 50 infants fed condensed milk there was an average weekly gain of 6 ounces as compared with 5.6 ounces in a control group. Growth, dentition and other developmental features were normal. Anemia developed in only 2 cases, and the number of infections was no greater than in a control group. Stringfield and Tobey [93] felt that condensed milk is of value as complementary feeding in the new-born and in athrepsia because of its ready digestibility, concentration, high carbohydrate content and consequent tendency to prevent acidosis. Evans [94] had excellent results from the use of condensed milk as a source of additional carbohydrate. A characteristic formula consisted of 2 ounces of condensed milk to 21 ounces of milk and 12 ounces of water, with a composition of 2.6 per cent fat, 2.8 per cent protein, and 6.8 per cent carbohydrate and a caloric value of 19 to the ounce. Infants showed an average weekly gain of 6.8 ounces during the first half year of life.

Of distinct value is Powers' [95] interesting concept of expressing the constitution of a number of milk mixtures commonly used in terms of

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91. Blatt, M. L., and Nichamin, S. J.: Infant Feeding in an Institutional Environment, J. Pediat. **2**:469 (April) 1933.

92. DeSanctis, A. G., and Craig, J. D.: Critical Clinical Study of Concentrated and Dried Infant Foods: Condensed Milk, Arch. Pediat. **48**:439 (July) 1931.

93. Stringfield, O. L., and Tobey, J. A.: The Advantages and Limitations of Condensed Milk in Infant Feeding, Arch. Pediat. **47**:769 (Dec.) 1930.

94. Evans, D. P.: New Method for Using Cane Sugar in Infant Feeding, J. M. Soc. New Jersey **29**:856 (Nov.) 1932.

95. Powers, G. F.: Comparison and Interpretation on Caloric Basis of Milk Mixtures Used in Infant Feeding, Am. J. Dis. Child. **30**:453 (Oct.) 1925.

the percentage of each energy producing nutrient, i. e., carbohydrate, fat and protein, in the total energy value of the food. Thus, he pointed out that the Czerny-Kleinschmidt and Moro butter-flour mixtures have about the same proportion of the total calories distributed among protein, carbohydrate and fat as has human milk. It is of particular interest that many of the most popular and commonly used formulas show the same caloric proportions of protein, carbohydrate and fat as do feedings of condensed milk.

*D. Concentrated Feedings.*—Relatively little that is new and of importance has been contributed in the past decade to the knowledge of concentrated feedings. Most articles have been devoted to the reporting of various new mixtures and the clinical results obtained with them. Probably the one outstanding contribution has been Rogatz'[96] work on peristolic function. Rogatz showed that with ordinary fluid feedings the stomach is pear-shaped, oval or elliptic and extends to or beyond the midline of the abdomen, with an air bubble varying greatly in size, shape and form in the upper portion of the stomach. When thick feedings are ingested, the stomach is much smaller (about one-third the size of the fluid form) with little or no air bubble, is circular or oval and barely reaches the midline. This is due to the fact that with thick feedings the stomach exerts markedly its peristolic function, which may be defined as the capacity of the wall of the stomach to surround or grasp its contents following the ingestion of food. Following fluid feedings the peristolic function of the stomach is at a disadvantage because of the inherent elusive nature of liquids which tend to travel away from a source of pressure, thus resulting in vomiting. Following thick feedings, however, the stomach can exert its peristolic function more successfully, for the thicker food is less mobile than fluids and offers a welcome resistance to the gastric musculature. This is of distinct importance in cases of vomiting and pylorospasm, and explains the results obtained with thick cereal feedings in these cases.

Schick[97] emphasized the fact that concentrated feedings should be reserved for children with definite indications, and that the normal infant does not need such a food. He listed as major indications such conditions as prematurity, nervous vomiting, pylorospasm, disorders of water balance and difficult feeding. Wisch[98] likewise pointed out that

96. Rogatz, J. L.: Roentgen-Ray Studies of Stomach Function, Am. J. Dis. Child. **28**:53 (July) 1924; Roentgen-Ray Studies of Stomach Function: Peristolic Function, ibid. **28**:69 (July) 1924; Peristolic Function: Its Practical Significance in Cases of Pylorospasm and Habitual Vomiting, ibid. **28**:582 (Nov.) 1924.

97. Schick, B.: Concentrated Feeding, Arch. Pediat. **42**:397 (June) 1925.

98. Wisch, L. J.: Some Observations on the Czerny-Kleinschmidt Butter-Flour Mixture, J. Indiana M. A. **17**:116 (April) 1924.

in such specific wasting diseases as syphilis it is folly to expect concentrated feedings to be successful without treating the primary condition.

From Germany have come a variety of concentrated and butter-flour formulas such as Jungwirth's [99] mixture of 130 Gm. of milk, 10 of flour, 10 of butter and 15 of sugar, all boiled down to 100 Gm., containing 260 calories. Pagorschelsky [100] had good results in 100 cases with the use of a protein milk-butter-flour mixture. This proved particularly valuable in the feeding of atrophic children recovering from diarrhea. Weil [101] used a concentrated buttermilk mixture composed of buttermilk to which 2 per cent of flour and 13 per cent of carbohydrate were added.

Chase [102] employed a formula containing 100 cc. of milk per kilogram of body weight with an equal amount of water to which from one-half to 1½ ounces of farina was added. This was cooked for from fifteen to thirty minutes, after which additional carbohydrate was supplied. The clinical results were excellent, as gains in weight were more than doubled when this formula was employed. Green and his associates [53] used a thick paste, made of lactic acid milk and farina, with good results in cases of vomiting and pylorospasm.

Aside from the actual thickening of formulas a good deal of modern tendency in feeding has been toward more concentrated formulas, as evidenced by the use of whole milk, either acidified or restored from evaporated milk. Additions high in carbohydrate resulting in more concentrated caloric mixtures have also been used with less and less trepidation, but this problem will be more fully discussed in the next section.

*E. Carbohydrates.*—The choice of the various carbohydrates, the increasing popularity of banana feeding and Gerstley's investigations deserve comment in a discussion of carbohydrates in modern infant feeding. Flood [103] investigated the rate of absorption of the different carbohydrates in the new-born. Of the monosaccharides, dextrose is, of course, the most easily absorbed, causing the quickest and highest increment in the blood sugar, followed in order by galactose and levulose. The ingestion of dextrose caused an increase of 40 per cent in the blood

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99. Jungwirth, G.: Erfahrungen mit Fettbreiernährung, Monatschr. f. Kinderh. **38**:500, 1928.

100. Pagorschelsky, H.: Ueber Eiweissmilch-Einbrenne, Ztschr. f. Kinderh. **40**:133, 1925.

101. Weil, H.: Concentrated Feeding with Buttermilk, Monatschr. f. Kinderh. **32**:40, 1926.

102. Chase, O. E.: Cereal Gruel Feeding, J. A. M. A. **84**:1173 (April 18) 1925.

103. Flood, R. G.: Rate of Sugar Absorption in New-Born, J. A. M. A. **82**: 1595 (May 17) 1924.

sugar level within twenty minutes, followed by a fairly rapid decline; galactose effected a fairly well maintained increment of 25 per cent in from forty-five to sixty minutes, and levulose caused an increase of 24 per cent in from sixty to seventy-five minutes with a prolonged descent. Of the disaccharides, lactose resulted in a fairly well sustained increase of 24 per cent in fifty minutes; sucrose showed only a 15 per cent increase in from eighty to ninety minutes, and maltose effected a 42 per cent increment, which was fairly well maintained, in from sixty to seventy-five minutes.

Flood [104] pointed out that the absorption of the polysaccharides which are used in infant feeding depends on two factors: (1) the speed and ease with which they are hydrolyzed into their component monosaccharides, and (2) the specific rates of absorption of these monosaccharides. Those that are absorbed slowly reach the colon and are susceptible to bacterial action. The sugars used in infant feeding may then be divided into three classes:

1. Sugars, such as corn syrup and dextrimaltose, which are hydrolyzed to dextrose only. These sugars are noted for their ease of absorption and lack of laxative action.

2. Sugars, such as lactose and sucrose, which are hydrolyzed to dextrose and to a monosaccharide other than dextrose, thus being slowly or not entirely absorbed, and consequently exerting (theoretically) a laxative action.

3. Sugars such as the banana, which contains a mixture of preformed dextrose and fructose, sucrose and starch, thus exhibiting both ease of absorption, because of the free dextrose, and a slight laxative action.

However, this moderate indictment of lactose has not gone unchallenged. Gerstley and his associates [105] performed an extremely pains-

104. Flood, R. G.: Selection of Sugars in Infant Feeding, Arch. Pediat. **42**:50 (Jan.) 1925.

105. Gerstley, J. R.; Wang, C. C.; Boyden, R. E., and Wood, A. A.: The Influence of Feeding on Certain Acids in the Feces of Infants: I. A Comparison of the Effects of Breast Milk and Modified Cow's Milk on the Excretion of Volatile Acids, Am. J. Dis. Child. **35**:580 (April) 1928; II. The Effect of an Excess of Lactose in Breast Milk and in Modified Cow's Milk, with Observations on a Parenteral Infection, ibid. **36**:289 (Aug.) 1928. Gerstley, J. R.; Wang, C. C., and Wood, A. A.: III. A Comparison of the Effects of Breast Milk and of Whole Cow's Milk on the Excretion of Volatile Acids and of Lactic Acid, ibid. **39**:487 (March) 1930; IV. The Effect of an Excess of Lactose in Breast Milk and in Whole Cow's Milk on the Excretion of Volatile Acids and of Lactic Acid, ibid. **39**:729 (April) 1930. Gerstley, J. R.: V. Clinical Considerations, ibid. **40**:27 (July) 1930; VI. Studies in Infant Nutrition: Lactose and a Maltose-Dextrin Preparation, J. A. M. A. **95**:1233 (Oct. 25) 1930.

taking and brilliant study related to the question of carbohydrate and lactose metabolism. Gerstley endeavored to investigate the etiology of nutritional disturbance in infancy. According to one school a diet containing excessive carbohydrate favored the development of diarrhea and nutritional upset because bacteria normally present in the intestines fermented the excess carbohydrate, causing irritation of the intestinal tract and leading to diarrhea and subsequent nutritional disturbance. The other school believed that the diarrhea was secondary, that cow's milk causes a primary injury to the intermediary metabolism of the body which leads to a secondary and more or less incidental diarrhea. Gerstley desired to test, both clinically and chemically, the validity of these respective theories. His mode of attack was: first, to determine the amount of acids in the stool on a diet low in carbohydrate; then to increase the carbohydrate content of the food, and then again determine the acids in the stool, with the obvious expectation of finding that the increased amount of carbohydrate would lead to increased intestinal fermentation and higher acid output. Gerstley ably summarized the work of his group. The stools of babies fed whole cow's milk are of greater weight and contain a greater amount of lower fatty volatile acids and their salts and a somewhat lower content of lactic acid and lactates than do the stools of breast-fed infants, but the hydrogen ion concentration (or true acidity) of the former is invariably less than that of the latter; that is, the stools of infants receiving cow's milk always show a $p_H$ more toward the alkaline side. (Gerstley reported the average $p_H$ for the stools of breast-fed infants as 4.5, and for stools of bottle-fed infants as 6.4. Dods'[106] findings were: stools of breast-fed infants under 3 months of age, $p_H$ 5.5; from 3 to 8 months of age, $p_H$ 6; stools of infants receiving cow's milk, $p_H$ 7.5. Brown and Tisdall[107] found that the average hydrogen ion concentration of stools of breast-fed infants ranged from $p_H$ 4.7 to $p_H$ 5.1. All are in active agreement, however, that the stools of infants receiving cow's milk are more alkaline than the stools of infants receiving breast milk). When diarrhea develops in the breast-fed infant, there occurs an increased frequency of stools with a small increase in their weight. The excretion of volatile fatty acids is slightly increased, but there is little change in the $p_H$ value. When diarrhea develops in infants receiving cow's milk, there is a great increase in the weight of the stools and a corresponding rise in the volatile fatty acids and lactic acid and their salts, but again the $p_H$ value is only slightly affected. The diarrheal stools of the infant fed

106. Dods, L.: Observations on the Hydrogen Ion Concentrations and Flora of Infants' Stools, M. J. Australia **1**:510 (April 20) 1929.

107. Brown, A., and Tisdall, F. F.: Factors Involved in the Acidity of the Stools of Infants, New York State J. Med. **25**:152 (Feb. 6) 1925.

on cow's milk contain more volatile fatty acids than do those of the breast-fed infant only because of the increased mass of the stool; unit for unit, they contain less acid. The addition of lactose to breast milk causes little change in the excretion of acids, weight of the stools or $p_H$ value. The addition of lactose to cow's milk causes some change at first, but, strangely enough, as the amount of lactose added is raised to 12 per cent the $p_H$ value, lactic acid, volatile acids, weight of the stools and total titratable acidity, quantitatively if not qualitatively, approximate the conditions found in the stool of the breast-fed infant. Apparently the relation of lactose to protein thus obtained (4:1) is similar to that of breast milk, and is most favorable to intestinal processes.

It was believed that in diarrhea something other than the amount of carbohydrate in the diet is at fault. Then diarrhea developed in a child who had been receiving whole milk; the diarrhea was cured by the addition of 3 per cent and, later, 12 per cent of lactose to the milk (Skole [108] also successfully used lactose, in the form of $\beta$ lactose, although not in such large amounts, in the treatment of diarrheal conditions in infants). Finally, and fortuitously, alimentary intoxication, with all its symptoms except diarrhea, suddenly developed in a child who had been receiving whole milk with 12 per cent additional lactose. This, it was felt, was prime evidence in favor of the argument that nutritional disturbance is independent of the gastro-intestinal tract, that cow's milk causes a primary injury to the body metabolism whereas intestinal fermentation and diarrhea are secondary and incidental and that severe nutritional disturbance may develop without diarrhea. It was also felt that lactose per se does not deserve its somewhat maligned reputation, and that some other element of cow's milk is at fault, probably by delaying the absorption of carbohydrate,[71] thus permitting greater fermentation. The relation of lactose to the protein content is also highly important.

Brown and Tisdall [107] had previously arrived at much the same conclusions. They, too, investigated the factors involved in determining the acidity of the stools of infants and decided that the amount of acidity was a resultant of the relationship between the carbohydrate and the protein content of the food, the carbohydrate producing the lactic and volatile fatty acids and the protein tending to cause an alkaline reaction because of the liberation of ammonia. They felt that if an infant were fed protein milk with its low carbohydrate and high protein content for a sufficient length of time for the low sugar content to interfere with the nutrition of the intestinal cells, the carbohydrate would no longer be absorbed as well as usual and thus would reach the zone of fermenta-

108. Skole, S. M.: Lactose in the Treatment of Diarrhea and Vomiting in Infants, Arch. Pediat. **50**:395 (June) 1933.

tion and cause diarrhea. The addition of more carbohydrate to the diet might then stimulate the intestinal cells to normal function and thus check the diarrhea. Thus, the acidity of the stool depends on the ratio, in the intestinal zone of fermentation, of carbohydrate to protein and base. The amount of carbohydrate in the zone of fermentation is dependent, partially, on the state of health of the intestinal cells.

That additions high in carbohydrate may be safe is evidenced by Bunnemann's [109] work. His supplementary feedings, even during the first few weeks of life, consisted of whole milk with 17 per cent additional cane sugar. As was previously mentioned, Happ supplied 10 per cent additional carbohydrate to skimmed milk for complementary feeding of the new-born.

To return more specifically to the problem of lactose itself, both clinical and laboratory reports tend to uphold Gerstley's contention that its previous disfavor is by no means merited. Thus, Dennett and Craig [63] reported the following average weekly gains in four groups of routine feeding cases in which cane sugar, dextrimaltose, lactose and corn syrup had been given as additional carbohydrate: cane sugar, 4.98 ounces; dextrimaltose, 4.8 ounces; lactose, 7 ounces; corn syrup, 5.77 ounces. Barenberg and Abramson [110] studied the effects of large additions of lactose to the dietary of infants. Ten infants receiving from 12 to 15 per cent additional lactose in the milk had no diarrhea during the summer months and gained an average of 22¾ ounces a month as compared with 14¼ ounces for the controls. Six infants during the winter months gained an average of 16 ounces a month as compared with 10½ ounces for the controls. Diarrhea was present only with infections of the upper respiratory tract, and was no more frequent than in the control group. They concluded that lactose is neither an intestinal irritant nor a laxative. Jarvis [111] showed that animals receiving lactose had more living tissue, i. e., more solids and proteins, and less water and fat than animals receiving other carbohydrates. The defense for lactose rests with a fairly strong case.

Can one as yet draw definite conclusions from the work of Gerstley and the other investigators? It would seem that the amount of lactose or other carbohydrate in the infant's diet is not per se the major etiologic factor in diarrhea; the ratio of carbohydrate to protein content rather than the absolute quantity of carbohydrate is of greater impor-

109. Bunnemann, A.: Concentrated Sugar Milk Feeding, München. med. Wchnschr. **71**:1644 (Nov. 21) 1925.

110. Barenberg, L. H., and Abramson, H.: The Effect of Large Amounts of Milk Sugar on the Stools and Nutrition of Infants, Arch. Pediat. **47**:1 (Jan.) 1930.

111. Jarvis, B. W.: Milk Sugar in Infant Feeding, Am. J. Dis. Child. **40**:993 (Nov.) 1930.

tance; there is probably some other factor, possibly in cow's milk whey, that plays a great rôle.

The use of the banana as a source of carbohydrate in infant feeding has increased of late. Von Meysenbug [112] gave the following analysis: protein, less than 1 per cent; carbohydrate, 20 per cent; fat, negligible amount, and ash, 0.83 per cent. Scriver and Ross' [113] figures are comparable: protein, 1.3 per cent; carbohydrate, 22 per cent; fat, 0.6 per cent, and ash, 0.8 per cent. In addition to its high carbohydrate content, the banana is also a fairly good source of vitamins. In vitamin C content it is second only to orange juice. Grüninger [114] both cured and prevented scurvy in guinea-pigs by the use of the banana. It is an excellent source of vitamin A, a good source of vitamin B but deficient in vitamin D. It also has a high iron content. In clinical use it has proved highly satisfactory both in the form of ripe mashed banana [115] and as powdered banana.[116] Kahn [117] found that a formula containing 5 per cent of powdered banana changed the fecal flora to a gram-positive organism made up largely of Micrococcus ovalis, which is similar to the effect caused by pure fructose, possibly because fructose is the least rapidly absorbed of the monosaccharides and offers, therefore, a favorable medium for the growth of M. ovalis and gram-positive anaerobic intestinal flora.

Honey [118] has also had a moderate vogue in other countries. Muniagurria [118a] has given as high as 200 Gm. a day, safely, often effecting excellent gains in weight. Of but passing interest is the direct use of such monosaccharides as galactose [119] and dextrose,[120] instead of the polysaccharides or disaccharides for additional carbohydrates.

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112. von Meysenbug, L.: Experiences with Banana Feeding in Infants, Arch. Pediat. **45**:509 (Sept.) 1928.

113. Scriver, J. B., and Ross, S. G.: The Use of the Banana as a Food for Healthy Infants and Young Children, Canad. M. A. J. **20**:162 (Feb.) 1930.

114. Grüninger, U.: Ueber den Vitamin C- und D-Gehalt der Bananen, Arch. f. Kinderh. **84**:284 (July 20) 1928.

115. von Meysenbug, L.: The Banana as a Food for Infants and Children, New Orleans M. & S. J. **80**:180 (Sept.) 1927.

116. Haas, S. V.: Powdered Ripe Banana in Infant Feeding, Arch. Pediat. **48**:248 (April) 1931.

117. Kahn, B. S.: Effect of Banana Powder Feeding on Fecal Flora, Arch. Pediat. **50**:330 (May) 1933.

118. (*a*) Muniagurria, C.: Le miel des abeilles dans la diététique normale et thérapeutique du nourrisson, Bull. Soc. de pédiat. de Paris **29**:227 (April) 1931. (b) Comby, J.: Milk with Honey in Infant Feeding, Arch. de méd. d'enf. **29**:46 (Jan.) 1926.

119. Bischoff, G.: Die Wirkung der Galaktose auf den Gewichtsansatz des Saüglings, Monatschr. f. Kinderh. **48**:500 (Dec.) 1930.

120. Hertz, W.: Klinische Prüfung der Verwendbarkeit des Traubenzuckers in der Therapie der Durchfallserkrankungen der Säuglinge, Arch. f. Kinderh. **92**:1 (Nov. 28) 1930.

*F. Milkless Diets: Vegetable Milks.*—The use of so-called milkless diets or, more properly, vegetable milks, of which soy-bean preparations are the most popular, is a subject that merits a certain amount of discussion in view of the increasing popularity which they have enjoyed in the past half a dozen years. Milk prepared from the soy-bean is not an innovation in infant feeding. It has been widely used in China for many centuries. Ruhräh was the first in this country, in 1909, to advocate the feeding of soy-bean milk to infants. Of late, Hill [121] has been one of the chief advocates of soy-bean milk in cases of infantile eczema. Several French investigators also gave favorable reports on it. As Maillet [122] pointed out, it is of particular value only when the infant's disturbance is due to some intolerance to the protein of cow's milk. Ribadeau-Dumas and his associates [123] listed such conditions as eczema, allergic phenomena, anaphylactic reactions and other idiosyncrasies to cow's milk as indications for vegetable milk feeding. They pointed out that when infants are first given feedings of soy-bean, there is frequently an initial loss of weight owing to changes in water balance, as many of these children are hydrolabile. This lost weight is soon recovered. Tso [124] made several interesting studies on the use of soy-bean milk in the infant's diet. The clinical course of 6 infants fed soy-bean milk for from six to nine months was normal; their weight curves and development were up to the average. Tso showed that the absorption of nitrogen from soy-bean milk is somewhat less than from cow's milk. However, he pointed out that in soy-bean feeding the protein intake is at a high level, comprising in energy equivalents about 20 per cent of the total calories as compared with a protein caloric value of 8 per cent in breast milk and from 12 to 15 in cow's milk. The nitrogen retention was somewhat better on cow's milk, with a protein caloric value of 12.2 per cent, than on soy-bean milk, with a protein caloric

121. Hill, L. W.: Infantile Eczema with Special Reference to the Use of a Milk-Free Diet, J. A. M. A. **96**:1277 (April 18) 1931; Infantile Eczema, J. Pediat. **2**:133 (Feb.) 1933. Hill, L. W., and Stuart, H. C.: A Soy Bean Food Preparation for Feeding Infants with Milk Idiosyncrasy, J. A. M. A. **93**:985 (Sept. 28) 1929.

122. Maillet, M.; Yeu, L., and Yeu, K.: Le lait de soja dans l'alimentation des nourrissons, Bull. Soc. de pédiat. de Paris **30**:488 (Oct.) 1933.

123. Ribadeau-Dumas, L.; Mathieu, R., and Wilhemin, L.: De l'utilisation des protéines végétales dans l'alimentation des nourrissons, Nourrisson **18**:161 (May) 1930.

124. Tso, E.: A Vegetable Milk Substitute for North China, Am. J. Physiol. **90**:542 (Oct.) 1929; The Development of an Infant Fed Eight Months on a Soy-Bean Milk Diet, Chinese J. Physiol. **2**:33 (Jan.) 1928. Tso, E., and Chu, F.: The Nitrogen Metabolism in Infants on Graded Intake of Soy-Bean Milk Proteins, Proc. Soc. Exper. Biol. & Med. **28**:218 (Dec.) 1931.

value of 18.5 per cent. Tso [125] and others have shown that soy-bean preparations are much richer in vitamin B, and contain about the same amount of vitamin A as cow's milk. Vitamins C and D are lacking. The stools of infants receiving soy-bean milk are bulky, and the type of flora resembles that of the breast-fed baby.[126]

The chief differences in a comparison of the mineral contents of soy-bean milk and cow's milk is the far lower calcium, sodium and chlorine content of the former.[127] Its phosphorus content is adequate and it is rich in potassium and magnesium.[128] Soy-bean meal especially, but also soy-bean cheese and milk, proved to be highly effective in the regeneration of hemoglobin in rats made anemic by an ordinary milk diet.[129] All three preparations are especially rich in iron and copper, soy-bean meal containing about sixty times as much iron and forty times as much copper as cow's milk. In this country the deficiencies in mineral content of a dried preparation of soy-bean flour have been remedied by the addition of 4.5 per cent dicalcium phosphate and 1.3 per cent sodium chloride.

While the soy-bean has been the most widely used of the vegetable proteins, other substances have also had some attention, more particularly almonds and coconuts. Klein [130] reviewed the use of some of these substances and quoted Chapin,[131] who showed that almond milk has a relatively large amount of phosphorus and a low sodium chloride content and is therefore useful in conditions such as rickets and nephritis. Gestura and Bahia [132] reported good results with the use of coconut milk in 50 cases of nutritional disturbance, and Johns and his co-workers [133] had previously shown that coconut contains all the essen-

125. Tso, E.: A Comparison of the Nutritive Properties of Soy-Bean Milk and Cow's Milk, Chinese J. Physiol. **3**:353 (Oct.) 1929.

126. Rittinger, F. R., and Dembro, L. H.: Soy Bean (Vegetable) Milk in Infant Feeding, Am. J. Dis. Child. **44**:1221 (Dec.) 1932.

127. Tso, E.; Yee, M., and Chen, T-T.: The Nitrogen, Calcium and Phosphorus Metabolism in Infants Fed on a Soy-Bean Milk, Chinese J. Physiol. **2**:409 (Oct.) 1928.

128. Stearns, G.: Soy-Bean Flour in Infant Feeding, Am. J. Dis. Child. **46**:7 (July) 1933.

129. Adolph, W. H., and Kao, H. C.: Hemoglobin Building Properties of Soy-Bean Products, Chinese J. Physiol. **6**:257 (Aug.) 1932.

130. Klein, J.: Vegetable Milk in Infant Feeding, Arch. Pediat. **50**:205 (March) 1933.

131. Chapin, H. D.: The Use of Vegetable Milk in Children, Tr. Am. Pediat. Soc. **30**:160, 1918.

132. Gestura, M., and Bahia, A.: Coconut Milk in Nutritional Disturbances in Infants, Brasil-med. **46**:169, 1932.

133. Johns, C.; Funk, A., and Paul, M. S.: The Nutritional Value of Coconut Globulin, J. Biol. Chem. **37**:497 (April) 1919.

tial amino-acids. Popowa [134] believed that almond milk was of aid in the treatment of several children with exudative diathesis. The milk was made by allowing 150 Gm. of almonds to soak in a liter of water, and to the strained milk was added an equal volume of whey and additional carbohydrate. Oser,[135] however, felt that almond milk was not satisfactory as an infant food.

Soy-bean milk is probably adequate in taking care of the present restricted needs and use of "milkless diets."

*G. Special Group.*—Before proceeding to the discussion of mineral metabolism and vitamins, there remain a few subjects which do not fall into any definite category. Some are of but passing interest.

Gelatin has been one of the substances tried by several investigators in infant feeding. As has been seen previously, Kugelmass used a 6 per cent gelatin solution for complementary feeding in the new-born. Gelatin has also been incorporated in routine formulas by others. Perlman [136] felt that gelatinized milk was advantageous to infants. When small amounts, such as 1 drachm of powdered gelatin, were added to the day's formula, infants seemed to vomit less and to gain in weight more rapidly. Hess [137] fed infants for alternating periods of three or four weeks on milk containing 1 per cent of gelatin by weight and milk with egg yolk and orange juice of equal caloric value. Gains in weight were about the same for each feeding, averaging 6.44 ounces a week for the "gelatinized" feeding and 6.46 ounces a week for the egg yolk-orange juice periods, as compared with 5.78 ounces during control periods. However, the caloric intake during the control periods was not quite so high. The stools during the "gelatinized" feeding periods were foul and at times passed with difficulty. Elterich [138] reviewed the subject of gelatin feeding and showed that large amounts may be given without any apparent unfavorable results. He used 4 tablespoonfuls, containing 100 calories, daily, but could not effect any acceleration of growth or gain in weight; in fact, the average weekly gain during the control periods exceeded, by almost an ounce, the average weekly gain during the experimental periods. Lautz showed by experiments in vitro that gelatin markedly decreased peptic digestion.

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134. Popowa, A.: Ueber einige Indikationen der milchfreien Diät bei Säuglingen und jungen Kindern, Monatschr. f. Kinderh. **35**:416, 1927.

135. Oser, W.: Rohkost in der Säuglingsernährung, Schweiz. med. Wchnschr. **62**:91 (Jan. 23) 1932.

136. Perlman, H. H.: Comparative Value of Gelatin as an Accessory Food, Arch. Pediat. **45**:14 (Jan.) 1928.

137. Hess, J. H., and Chamberlain, I. M.: Gelatin Added to Diets of Artificially Fed Infants, J. A. M. A. **89**:1423 (Oct. 22) 1927.

138. Elterich, T. O.; Boyd, D. H., and Neff, A.: The Use of Gelatin as a Supplementary Food in the Infant's Dietary, Arch. Pediat. **47**:286 (April) 1930.

An interesting report, intimately connected with the question of "gelatinized" feedings and introducing colloid chemistry into the realm of infant feeding, came from France. Bentivoglio,[139] after a preliminary discussion of the necessity of diluting cow's milk and the disadvantages of this dilution, discussed the colloidal differences in the dispersion of casein in bovine and human milk, and showed that it is these differences that necessitate dilution of the cow's milk. In human milk the high percentage of albumin exerted a protective colloidal action on the casein. He felt that if a protective colloid (such as gelatin or acacia) were added to cow's milk, it would change the colloidal action of the casein and thus allow higher concentrations of milk in formulas. He used from 3.5 to 2 Gm. of acacia in 10 per cent solution, per hundred grams of milk. The higher amounts were used in the first month of life and then gradually diminished. By the use of this substance he was able to use a formula of three-fourths milk, the only dilution being by the gum solution, as early as the tenth day of life. Landsberger [140] conducted an experiment which has some connection with this colloid factor. He showed that the cleavage of tributyrin by lipase, obtained from an infant's stomach, occurred in vitro more quickly in whey of human milk than in whey of cow's milk, and felt that this was probably due to differences in colloidal substances, because when the wheys were freed of albumin, this difference in the time of fat-splitting disappeared.

Of passing interest is the report on the use of intarvin, a fat with an odd number of carbon atoms originally intended for diets for diabetic patients. Horowitz [141] added from 0.5 to 1.5 Gm. of an artificial fat from fatty acids having an odd number of carbon atoms (as a 6 per cent aqueous emulsion) per kilogram of body weight to the feedings. In 10 of 11 cases the gain in weight was distinctly greater when the infants were offered this supplemented diet.

Of distinct interest, however, is C. M. Davis' [142] experiment on self-selection of diets by infants. Three infants of weaning age were subjected to self-selection of diets for periods of from six months to a year. In her own words, "The results seemed at least equal to the best results obtained by commonly prescribed diets in growth, weight, bone development, musculature, general vigor and appearance of health and well-being." Further routine experiments included older children,

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139. Bentivoglio, G. C.: Comment on peut administrer aux nourrissons même très petits le lait de vache très faiblement dilué, Nourrisson **19**:22 (Jan.) 1931.

140. Landsberger, M.: Action of Gastric Lipase in Whey of Human and Cow's Milk, Ztschr. f. Kinderh. **39**:665, 1925.

141. Horowitz, P.: The Use of Odd-Carbon Fat in Infant Feeding, Arch. Pediat. **44**:293 (April) 1927.

142. Davis, C. M.: Self Selection of Diet by Newly Weaned Infants, Am. J. Dis. Child. **36**:651 (Oct.) 1928.

with the happiest of results for both subjects and observers. If this method can only be impressed on mothers met in private practice, possibly that bugbear of modern pediatrics—anorexia—can be done away with.

Some mention should be made of several reports concerning the relative superiority of certified milk over pasteurized milk. Lewis [143] fed one group of 122 infants certified milk and a second group of 112 infants pasteurized milk. Both milks were boiled for two minutes before use. The first group showed distinctly better gains in weight in all three age groups considered: from birth to 3 months, from 3 to 6 months, and from 6 to 9 months of age. Not only was the average gain in weight superior, but the general development and activity of the first group seemed better, and whatever rickets occurred was milder. Ladd and his associates [144] also found that certified milk caused a considerably greater average gain in weight than pasteurized milk alone or pasteurized milk with cod liver oil and orange juice. Both Lewis and Ladd felt that this superiority was due to the fact that certified milk is of better quality owing to more exact and scientific care and feeding of cows.

# Pediatric Biographies

## JOHN ABERCROMBIE
1780–1844

### A NOTE ON THE HISTORY OF TYPHOID FEVER IN CHILDREN

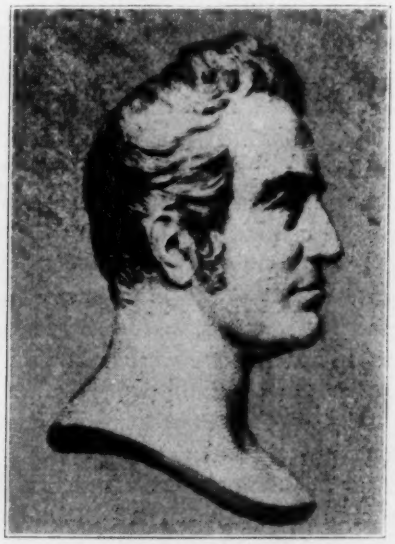
JOHN RUHRÄH, M.D.
BALTIMORE

John Abercrombie (1780-1844) was born at Aberdeen and was educated there in the public schools and at Marischal College; he went to Edinburgh for his medical training and received his degree in 1803. He first became interested in the mental side of medicine, and his inaugural dissertation was entitled "De fatuite alpina." After a year at St. George's in London, he returned to Edinburgh, where he became attached to the public dispensary and established himself in practice. In both of these fields he became familiar with the life of the poor. He had an office in the poorer part of the town and he divided the district into sections and gave them over to his students, keeping the general supervision for himself. He was a sharp observer and kept records of all cases of interest. Many of these were elaborated into papers, which for the most part appeared in the *Edinburgh Medical and Surgical Journal* from 1816 to 1824. From these papers he made two books of unusual value, which repay reading today. Both were published in 1828, and the titles are "Pathological and Practical Researches on Diseases of the Brain and Spinal Cord" and "Pathological and Practical Researches on Diseases of the Stomach, the Intestinal Canal, the Liver and the Other Viscera of the Abdomen." Both are storehouses of clinical knowledge. When James Gregory died in 1821, Abercrombie became one of the foremost consultants in Scotland. He became a licentiate of the College of Physicians in 1823 and the next year was made a fellow. He was also given the honorary title of physician-in-ordinary to the kings of Scotland. In 1830 he published "Inquiries Concerning the Intellectual Powers and the Investigation of Truth;" in 1833, "The Philosophy of the Moral Feelings," and in 1835, a volume of essays and tracts, chiefly on moral and religious subjects. He was much more successful as a clinician than as a moral philosopher.

Abercrombie reported a case of typhoid fever with perforation of a typhoid ulcer in a young girl. The report is to be found on page 286 of his "Pathological and Practical Researches on Diseases of the Stomach, the Intestinal Canal, the Liver and Other Viscera of the Abdomen" (1830).

XII.—THE DISEASE OCCURRING IN CONTINUED FEVER, WITH ULCERATION

CASE XCIV.—A girl, aged 9, was seen by Dr. Alison in December, 1819, affected with the usual symptoms of contagious fever, which was very prevalent in a narrow and crowded lane where she resided, and had affected a person in an adjoining room. From the commencement of the disease, she had diarrhoea, with griping, and considerable tenderness of the abdomen; and the evacuations were thin, feculent, and of a healthy appearance. These symptoms continued, with frequent pulse and foul dry tongue, till about two days before her death, when the diarrhoea suddenly subsided, and was succeeded by violent pain, acute tenderness of the abdomen, and every symptom of peritoneal inflammation. The duration of the case was about three weeks. I am indebted to Dr. Alison for



JOHN ABERCROMBIE

the above outline of it, and for an opportunity of being present at the examination of the body.

*Inspection.*—There was considerable peritoneal inflammation, especially on the ileum, where there was extensive adhesion, with considerable deposition of flocculent matter. The intestine was also seen to be in several places perforated by small ulcerations, through which feculent matter had escaped into the cavity of the peritoneum. The ileum being laid open, discovered a most extensive tract of disease on its inner surface, the mucous membrane being extensively eroded, and in many places completely destroyed, by round well defined ulcers, many of them as large as a shilling. This state of disease extended over the greater part of the ileum, and in several places, its coats were considerably thickened. It contained a considerable quantity of fluid feculent matter, which was quite healthy in its appearance. The higher parts of the small intestine were healthy, and contained a small quantity of a dark green viscid fluid, like inspissated bile. The colon was collapsed, and externally healthy; internally, there were in several places, espe-

cially on the left side, patches of redness on its mucous membrane, but without any appearance of ulceration; it contained only a small quantity of healthy mucus. The other viscera were healthy.

As in this case there was every reason to believe that the original disease was contagious fever, the affection of the bowels may perhaps be considered as symptomatic.

# Pediatrics in Art

## THE DOCTOR AND THE POOR PATIENT

JOHN RUHRÄH, M.D.
BALTIMORE

In the eighteen hundred and sixties and until the gay nineties every household which was supposed to be strictly up to date possessed one of John Rogers' groups, which covered a wide range of subjects. The one in the illustration has a particular interest, for Dr. Willard Parker, the celebrated New York surgeon for whom the Willard Parker Hospital in New York was named, served as a model for the doctor. The dressing gown and smoking cap, in those days familiar enough, seem strange today.

**A John Rogers group, showing a doctor and a poor patient. The doctor is Willard Parker.**

Dr. Parker was a remarkable character, and a sketch of his life, together with the diary he kept while studying in Paris, edited, appeared not long ago in the *Annals of Medical History*. Dr. Parker was one of the first to operate for appendicitis.

John Rogers was born in Salem, Mass., in 1829. He worked in a machine shop for ten years, then studied in Rome, and later was a draughtsman in Chicago, but owing to the success of a group, "The Checker Players," he devoted himself to making similar groups and also to making life-sized portraits, including one of Lincoln. He died in New Haven in 1904.

# News and Comment

## SOCIETY NEWS

**Atlantic City Session of the American Medical Association.**—The Section on Pediatrics of the American Medical Association has appointed the following section exhibit committee for the Scientific Exhibit at the Atlantic City session of the American Medical Association and the Canadian Medical Association: F. Thomas Mitchell, Memphis, Tenn., chairman; Abraham Levinson, Chicago; Walter B. Stewart, Atlantic City, N. J., and Allan Brown, Toronto, Canada. Application for space must be made by Feb. 25, 1935. Application blanks may be obtained from any of the members of the committee or from the Director, Scientific Exhibit, 535 North Dearborn Street, Chicago.

## UNIVERSITY NEWS

Dr. Guillermo Arce has been called to the chair of pediatrics at the University of Santiago.

## PERSONAL

Secretary of Labor Perkins announced the promotion of Dr. Martha M. Eliot, director of the child and maternal health division of the Children's Bureau, to assistant chief of this bureau. The position is a civil service one. Dr. Eliot will have special responsibility for the development of the child and maternal health program of the Children's Bureau, including the study of the relationship of mental health to the prevention of delinquency.

Dr. Eliot joined the staff of the Children's Bureau in 1924 and has just completed her tenth year as director of the division of child and maternal health. She is a trustee of Radcliffe College, a member of the American Pediatric Society, chairman of the Child Health Committee of the American Academy of Pediatrics and consultant on child health of the Association of State and Provincial Health Officers.

Dr. Martha M. Eliot has been made assistant chief in charge of the medical work of the Children's Bureau of the U. S. Department of Labor.

Dr. Eliot is well known for her contributions to preventive pediatrics and likewise for her scientific investigations in the problems of rickets.

She graduated from Johns Hopkins University School of Medicine in 1918 and served during her internship and resident years at the Peter Bent Brigham Hospital, Boston; St. Louis Children's Hospital; Massachusetts General Hospital, Boston, and the New Haven Hospital. She is now associate clinical professor in pediatrics at Yale University.

Her society connections include the American Pediatric Society, the American Academy of Pediatrics, the American Medical Association, the Massachusetts Medical Association and the Connecticut Medical Association.

## DEATHS

Dr. H. Dorlencourt of the Hospice des enfant's-assistés died recently.

# Abstracts from Current Literature

## Pathology, Bacteriology and Biochemistry

THE EFFECT OF REMOVAL OF THE SYMPATHETIC CHAINS AND OF THE COELIAC GANGLIA ON GASTRIC ACIDITY. MADELAINE R. BROWN, Am. J. Physiol. **105**: 399 (Aug.) 1933.

There was no demonstrable change in either free or combined gastric acidity following removal of sympathetic chains or celiac ganglions of cats. Since the stomach may function automatically in the absence of extrinsic nerves, it is not probable that degeneration of the autonomic nervous system would lead to either hyperchlorhydria or achlorhydria.

NOURSE, Cleveland.

STUDIES IN THE NORMAL HUMAN WHITE BLOOD CELL PICTURE: I. VARIATIONS IN RECUMBENT BASAL SUBJECTS AND IN INDIVIDUALS WITH CHANGE OF POSTURE. EDGAR JONES, D. J. STEPHENS, HARRIETT TODD and JOHN S. LAWRENCE, Am. J. Physiol. **105**:547 (Sept.) 1933.

Studies were made on normal adults. White and differential blood counts were taken at fifteen minute intervals over a period of five hours. Under basal conditions the total numbers of white blood cells showed only slight variations. When short periods of activity were allowed, no significant changes were observed. Likewise, change of posture from the recumbent to the standing position did not influence the blood counts. Schilling differential counts failed to reveal any fluctuations in the "stab" forms, indicating that the physiologic variations which did occur were probably not due to changes in activity on the part of the bone marrow.

NOURSE, Cleveland.

STUDIES IN THE NORMAL HUMAN WHITE BLOOD CELL PICTURE: II. THE EFFECT OF DIGESTION ON THE WHITE BLOOD CELLS. JOHN S. LAWRENCE, D. J. STEPHENS and EDGAR JONES, Am. J. Physiol. **106**:309 (Nov.) 1933.

White blood cell counts were taken for fifteen normal adults at fifteen minute intervals for a period of five hours following a large meal. An increase in the total number of white blood cells in the peripheral blood was shown to occur in the majority of the subjects, but the increase was not of sufficient magnitude to be of clinical importance.

NOURSE, Cleveland.

IS THERE A SPECIFIC DIURETIC HORMONE IN THE ANTERIOR PITUITARY? B. O. BARNES, J. F. REGAN and J. G. BUENO, Am. J. Physiol. **105**: 559 (Sept.) 1933.

Marked diuresis such as occurs in normal animals following the injection of extracts from the anterior lobe of the pituitary gland was not observed in thyroidectomized dogs. It is suggested that the diuresis is a result of stimulation of the thyroid.

NOURSE, Cleveland.

THE THEBESIAN VESSELS AS A SOURCE OF NOURISHMENT FOR THE MYOCARDIUM. ANNE BOHNING, KENNETH JOCHIM and LOUIS N. KATZ, Am. J. Physiol. **106**:183 (Oct.) 1933.

The authors conclude from their experiments that in the functioning heart the thebesian vessels form a pathway by which blood can be conveyed from the chambers of the heart to the intramural coronary vessels. Under normal conditions, when the coronary arteries are occluded, enough blood may find its way into the coronary capillaries to aid appreciably in the nourishment of the heart.

NOURSE, Cleveland.

THE INTERRELATION OF A GONOTROPIC HORMONE AND VITAMIN A. S. B. D. ABERLI, Am. J. Physiol. **106**:267 (Nov.) 1933.

A placental extract standardized in regard to its production of mucoid vaginal cells was incapable of producing any effect on the vaginal cells of rats suffering from vitamin A deficiency. The mitotic figures present in the vaginas of rats on the diet deficient in vitamin A showed that the cells were viable and capable of proliferation. That the hormone was effective in the animals with vitamin A deficiency was shown by the distention of some of the uteri. The control animals invariably responded to the hormone by the production of mucoid vaginal cells. This shows that vitamin A deficiency was responsible for preventing the characteristic action of a hormone.

NOURSE, Cleveland.

STUDIES IN THE REGULATION OF GASTRIC ACIDITY: I. THE INFLUENCE OF ACID ON THE SECRETION OF HYDROCHLORIC ACID BY FUNDIC POUCHES AND BY THE WHOLE STOMACH. CHARLES M. WILHELMJ, IRWIN NAIGUS and FREDERICK C. HILL, Am. J. Physiol. **106**:381 (Nov.) 1933.

This study is an investigation of one concept of the regulation of gastric acid, namely, the theory that the concentration of hydrogen ions of the gastric contents determines the amount of acid secreted by the parietal cells. It was found that solutions of hydrochloric acid ranging in strength from 0.024 to 0.173 per cent normally do not inhibit the secretion of acid in fundic pouches or in the whole stomach of dogs. The same is true for nitric and sulphuric acids alone or in combination with sodium chloride.

NOURSE, Cleveland.

THE OUTPUT OF EPINEPHRIN FROM THE ADRENAL GLANDS DURING ANAPHYLACTIC SHOCK. MILTON B. COHEN, J. A. RUDOLPH, P. WASSERMAN and J. M. ROGOFF, Am. J. Physiol. **106**:414 (Nov.) 1933.

Anaphylactic shock in dogs sensitized with horse serum does not cause any significant alteration in the rate of secretion of epinephrine from the suprarenal glands.

NOURSE, Cleveland.

## Nutrition

EFFECT OF OVERDOSES OF IRRADIATED ERGOSTEROL, ADMINISTERED FOR APPROXIMATELY TWO MONTHS, ON THE COMPOSITION AND STRUCTURE OF THE BONES OF RATS. J. H. JONES and G. M. ROBSON, Am. J. Physiol. **102**:338 (Feb.) 1933.

The bones of rats which were given a sufficient amount of viosterol to produce death after a period of from six to eight weeks showed marked porosity due to the removal of organic as well as inorganic matrix—apparently, as the result of osteoclastic activity which was shown by histologic studies. The bones were soft and fragile; chemically there was a relative decrease in the ash content, which was apparently caused by the growth of osteoid tissue following the absorption of the bone matrix.

NOURSE, Cleveland.

THE SIGNIFICANCE OF THE HYPERTROPHY AND HYPERPLASIA OF THE PARATHYROID GLANDS IN RICKETS AND OSTEOMALACIA. RUSSELL M. WILDER, GEORGE M. HIGGINS AND CHARLES SHEARD, Ann. Int. Med. **7**:1059 (March) 1934.

Erdheim first suggested the relationship between the function of the parathyroid glands and rickets. His conclusions preceded the discovery of vitamin D and were based on the observation that in osteomalacia and rickets the parathyroid glands were enlarged. Others found a constant hyperplasia and hypertrophy of the parathyroid glands in chickens deprived of vitamin D.

The authors performed a series of experiments on chickens to determine whether enlargement of the parathyroid is an index of increased parathyroid function. Chickens which had been deprived of vitamin D were selected. Parathyroid hormone was administered to them, in the belief that if hypertrophy and hyperplasia were the result of vitamin D deficiency, the hormone given by injection would prevent the development of these abnormalities.

As a result of this work, it was concluded that deprivation of vitamin D results in hypertrophy and hyperplasia of the parathyroid glands, but only if the deprivation of vitamin D is insufficient to cause rickets. The administration of parathyroid hormone parenterally prevents hypertrophy and hyperplasia in minor degrees of vitamin D deficiency, but in extreme degrees of such deficiency the hormone restricts but does not prevent hypertrophy and hyperplasia of the glands.

Other observations led the authors to believe that the parathyroid glands have the power to accelerate the rate of supply of their product so that the organism might be able to withstand periods of relative vitamin D deficiency, which otherwise would result in rickets or osteomalacia. This mechanism is not adequate in extreme degrees of vitamin D deficiency. It is believed that this action depends on the effect of the hormone on the tissue, which it sensitizes or conditions so that normal function can take place with less vitamin than would otherwise be necessary.

READING, Galveston, Texas.

AVITAMINOSIS: XVI. PRODUCTION OF GASTRIC ULCERS IN THE ALBINO RAT AS A RESULT OF THE SPECIFIC INFLUENCE OF DEFICIENCY OF VITAMIN B. BARNETT SURE and HARVEY S. THATCHER, Arch. Path. **16**:809 (Dec.) 1933.

Gastric ulcers were produced in albino rats as a result of a specific deficiency in vitamin B uncomplicated by inanition. Photomicrographs of the ulcers are reproduced. The rats with ulcers were those suffering from a deficiency of vitamin B and not from a deficiency of both vitamins B and G. It is suggested that vitamin B therapy may be indicated in human gastric ulcer.

LEMASTER, Evanston, Ill.

INFANT FEEDING. WILLIAM WILLIS ANDERSON, Arch. Pediat. **50**:519 (July) 1933.

This article offers nothing of particular value.

CALCIUM AND PHOSPHORUS STUDIES: VII. THE EFFECTS OF VARIATIONS IN DOSAGE OF CALCIUM AND PHOSPHORUS IN THE DIET ON THE CONCENTRATIONS OF CALCIUM AND INORGANIC PHOSPHORUS IN THE SERUM AND ON THE HISTOLOGY AND CHEMICAL COMPOSITION OF BONES OF RATS. DAVID H. SHELLING, DOROTHY E. ASHER and DEBORAH A. JACKSON, Bull. Johns Hopkins Hosp. **53**:348 (Dec.) 1933.

The effects of parathyroid extract on the bones of young rats vary with the dose of the hormone and the calcium and phosphorus content of the diet. In animals fed the stock diet, parathyroid extract administered in amounts as high as 10 units daily for a rat weighing from 50 to 60 Gm. produces hyperostosis, while in amounts of 20 units it induces osteodystrophia fibrosa. In animals fed rachitogenic diets, the parathyroid extract fails to prevent rickets, but stimulates osteoblastic activity and trabecular proliferation. In rats fed "border-line" rachitogenic diets, the extract stimulates cartilaginous proliferation as well as increased trabeculation. In animals fed optimal calcium-high phosphorus diets, fibrosis occurs with a relatively lower dose of the extract than on the stock diet.

Injections of parathyroid extract evoke moderate hypercalcemia and hyperphosphatemia in the rat; the hyperphosphatemia, however, depends on the dose of the hormone and the intake of phosphorus. In animals fed the stock diet, larger doses of the extract increase the serum inorganic phosphorus nearly 100 per cent, but in those fed the rachitogenic diet the serum inorganic phosphorus remains at rachitic levels. In one group of animals fed a diet low in calcium and phosphorus, which were given injections of 10 units of the extract daily, the

serum calcium was 5.3 and the inorganic phosphorus 14.3 mg. per hundred cubic centimeters. In spite of the hypocalcemia and hyperphosphatemia, there was no clinical evidence of tetany.

Analysis of the percentages of ash, calcium and phosphorus of the dry extracted femurs indicated certain dissimilarities between the osseous hypercalcification due to feeding vitamin D and that induced by the parathyroid extract. The former increased the deposition of lime salts in the existing organic matrix so that the ratio of ash to organic matter was increased, whereas the parathyroid extract first stimulated trabeculation, and the calcification that followed did not exceed the production of organic matrix; hence the ratio of ash to organic residue was not increased, and was actually diminished if fibrosis and osteoid tissues were also present.

The injection of large doses of parathyroid extract into young suckling rats resulted in proliferation of cartilage, delayed ossification of the epiphyseal centers and decalcification and fibrous replacement of the metaphyses, but not typical rickets. Large doses of the extract produced lesions of the skin resembling scleroderma in 10 day old suckling rats. These lesions, however, were absent in older rats so treated. Metastatic calcification occurred only when the doses of the extract were very high and when the bones were markedly decalcified.

Certain discrepancies in the literature as to the effect of parathyroid extract on the rat are discussed, and the divergent results are explained as possibly due to variation in: (1) the age of the animals, (2) the dietary calcium and phosphorus and (3) the dose of the hormone.

AUTHORS' SUMMARY.

CAUSES FOR POOR NUTRITION OTHER THAN THOSE WHICH ARE PURELY PHYSICAL. C. ANDERSON ALDRICH, Illinois M. J. **65**:51 (Jan.) 1934.

Causes which are not strictly physical exert a profound effect on nutrition in all classes of society. Aldrich groups them under three main headings: (1) psychogenic anorexia, (2) poor habits and (3) poverty. He points out that good development depends less on adequate caloric intake than on qualitative considerations in the diet. To avoid psychogenic anorexia and insure adequate nutrition, the technic of feeding children must be carefully controlled so that their natural desires are given a free rein, and the diet should contain only such foods as are qualitatively adequate. The other habits of life which most directly influence nutrition are those of sleep, play and bowel control. Poor habits of sleep lead to fatigue, susceptibility to disease, anorexia and other nervous symptoms which hinder the attainment of optimum nutrition. Play should have its regular time and place in the child's life, because it is of utmost importance in his developmental welfare. Regular habits in bowel elimination are of utmost importance to nutrition because of the bad results of persistent stasis on appetite and digestion. The early diet should be so arranged that involuntary evacuations will be easy and fairly frequent; one should wait until the child can cooperate intelligently before attempting to teach voluntary control. Under the pall of poverty the new-born babe is already poorly nourished. In the later months the same condition holds true, even if the babies are breast fed, because breast milk can contain only those factors stored or eaten by the mothers. If the babies raised in poverty are artificially fed, the situation becomes more acute, unless two factors enter to protect the child—charity and education. Inadequate heating of houses, overcrowded living conditions, unsanitary arrangements, lack of sunshine, poor personal hygiene and filth must be added to the unfavorable factors due to or fostered by poverty.

BARBOUR, Peoria, Ill.

PHYSICAL DEFECTS COMMONLY FOUND WHICH INFLUENCE NUTRITION AND WHICH ADMIT OF CORRECTION. A. H. PARMELEE, Illinois M. J. **65**:53 (Jan.) 1934.

Some of the common physical defects which influence nutrition are: (1) physical defects which mechanically interfere with the taking of food, such as

carious teeth, improper occlusion and nasal obstruction and (2) physical defects which directly or indirectly are responsible for a poor appetite, such as nasopharyngeal discharges, nasal obstruction, defects of vision, defects which interfere with normal locomotion and infections. Parmelee points out that the correction of these defects alone may not be sufficient to effect an improvement in nutrition at this stage, for the disturbed nutrition, if of long standing, has caused damage to many tissues and organs of the body.

BARBOUR, Peoria, Ill.

THE INFLUENCE OF ACUTE AND CHRONIC CONDITIONS UPON NUTRITION. STANLEY GIBSON, Illinois M. J. **65**:54 (Jan.) 1934.

Gibson emphasizes the fact that malnutrition is not a disease but a symptom. The importance of infections of the upper respiratory tract, heart disease, tuberculosis and other diseases as etiologic factors are stressed.

BARBOUR, Peoria, Ill.

AN UNUSUAL INFANT DIET AND ITS QUESTIONABLE EFFECT UPON DENTITION. R. C. WILLETT, Internat. J. Orthodontia **20**:432 (May) 1934.

Willett reports three cases of children with abnormalities of the calcified tissues of the teeth and histories of nutritional disturbances in the first year. The first case is that of a male infant who, after several diets were tried, was given home brew (beer) exclusive of other nourishment for three months (from the seventh to the tenth month). During this time he gained 8 pounds (3,628.73 Gm.)—the first material gain in weight since his birth. When his deciduous teeth erupted they were devoid of enamel, and later the permanent teeth were of peculiar texture, but not hypoplastic. There was no evidence of dental caries at the age of 6 years and 10 months. He has suffered no serious illnesses, and other than having a mentality slightly below that of the average child of his age appears to be normal.

The second case is that of a boy who experienced some nutritional disturbances in the first half of his first year and who was artificially fed. His deciduous teeth erupted at about the fourth to the fifth month; they were entirely devoid of enamel. There is a history of this condition in the paternal great-grandmother, the paternal grandmother and the father. This child also had marked hypoplasia of the first permanent molar teeth; the remaining permanent teeth that have erupted (seventeen) appear to be normally formed.

The third case is that of a brother of the second patient, with teeth of normal form, but having a peculiar bluish translucent enamel that casts a lighter roentgenographic shadow than those with normal enamel. Two sisters in this family possessed normal teeth.

NOYES, Chicago.

A STUDY OF THE EFFECT OF PRESERVING METHODS ON HUMAN MILK. WALTER H. EDDY and STEWART G. MORRIS, J. Pediat. **4**:208 (Feb.) 1934.

Frozen human milk and dehydrated human milk were tested for the content of vitamins. Freezing was found to be a satisfactory method for preserving human milk, since the significant vitamins of human milk (A, B and G) were little affected by the process. Dehydration caused lowering of the vitamin A content. Human milk is considered an unreliable source of vitamins C and D.

LEMASTER, Evanston, Ill.

ANALYSIS OF SAMPLE DIETS AND DAILY HABITS OF 206 CHILDREN FROM DATA SUPPLIED BY PARENTS. WILLIAM PALMER LUCAS, HELEN BRENTON PRYOR and SAXTON T. POPE, J. Pediat. **4**:221 (Feb.) 1934.

The diets and daily habits of two hundred and six children were studied. The total number of calories and the percentage of protein, carbohydrates and fat in the diets of the children of various ages are presented statistically. Children of

the linear type were usually given a diet high in fat by their parents. Because of this fact and because anorexia was found so frequently in children of the linear type, the authors advocate a diet low in fat for slenderly built children. Information on ten items pertaining to the daily habits of the children was obtained and analyzed.

LEMASTER, Evanston, Ill.

THE PROPHYLACTIC USE AND TOLERANCE OF HALIVER OIL WITH VIOSTEROL IN PREMATURE AND WEAKLING INFANTS. EARL W. MAY and THELMA M. WYGANT, J. Pediat. **4**:226 (Feb.) 1934.

Forty premature and ten weakly infants given varying amounts of halibut liver oil with viosterol 250 D were observed for several months. The calcium and phosphorus values of the blood serum, the gain in weight, roentgenographic and clinical evidence of rickets, tolerance for various doses of halibut liver oil and determinations of hemoglobin were studied. When it was found that the combination of haliver oil and viosterol was well tolerated, a daily dosage of from 18 to 24 drops (3 drops per minim) was used. This preparation allows an abundance of vitamins A and D to be given in small doses which are easily taken by even the smallest premature infants.

LEMASTER, Evanston, Ill.

A STUDY OF BREAST MILK FAT. P. E. ROLLER, J. Pediat. **4**:238 (Feb.) 1934.

An investigation was made to determine the composition of the water-insoluble fatty acids found in breast milk fat. The results of other chemical and physical tests on breast milk fat are also tabulated.

LEMASTER, Evanston, Ill.

THE EFFECT OF SMALL QUANTITIES OF BREAST MILK, LIVER EXTRACT, IRON AND COPPER, RESPECTIVELY AND IN COMBINATIONS, UPON THE IRON BALANCE OF ARTIFICIALLY FED INFANTS. SIEGFRIED MAURER, JOSEPH GREENGARD, W. L. CURTIS and CESSA KLUVER, J. Pediat. **4**:356 (March) 1934.

The authors draw the following conclusions after testing the metabolism of seventeen infants: 1. Nine of twelve artificially fed infants showed negative iron balances, the more severe anemias having been present in the group which had a rather high negative balance. 2. A small quantity of breast milk greatly facilitated the absorption of iron from the gastro-intestinal tract. 3. In the artificially fed infants studied, copper and iron showed no more effect on the iron balance than iron alone. 4. The feeding of liver extract and iron resulted in the most marked improvement in the iron balance of the artificially fed infants studied, whether the iron was derived from inorganic salts or from hemoglobin.

LEMASTER, Evanston, Ill.

CONSTIPATION IN BREAST FED INFANTS CAUSED BY ANORECTAL FISSURE. DON F. CATHCART, J. Pediat. **4**:366 (March) 1934.

Two cases of constipation due to anorectal fissure in breast fed infants, aged 4 months and 7 months, respectively, are reported. One infant showed rapid improvement after the use of a bland ointment applied locally and mineral oil given by mouth. The other infant improved slowly under treatment consisting of silver nitrate applied locally and mineral oil given orally.

LEMASTER, Evanston, Ill.

RICKETS IN RATS BY IRON FEEDING. J. F. BROCK and L. K. DIAMOND, J. Pediat. **4**:442 (April) 1934.

The addition of ferric chloride to a nonrachitogenic diet caused the appearance of rickets in white rats. Increasing the phosphorus content of the diet prevented

the production of rickets in animals on the ferric chloride diet. The assumption is that iron enters into chemical combination with phosphorus in the intestinal tract to form compounds which are relatively difficult to absorb. Other compounds of iron besides ferric chloride also produced rachitic changes in rats, when given with a normal diet. It is conceivable that the continual use of large doses of iron by a child on a diet somewhat low in phosphorus might lead, over a prolonged time, to the same result as a diet low in phosphorus.

LEMASTER, Evanston, Ill.

A COMPARISON OF THE NUTRITIONAL AND GROWTH VALUES OF CERTAIN INFANT FOODS. C. T. WILLIAMS and A. O. KASTLER, J. Pediat. **4**:454 (April) 1934.

Eighty infants were divided into four groups and observed for six months. Group 1 was breast fed entirely. Group 2 received a formula of certified milk, water and lactose. Group 3 was fed a formula of evaporated milk, water and lactose, and group 4 received a formula of evaporated milk, water, corn syrup and lactic acid.

The breast fed infants gained weight more rapidly than the bottle fed infants. A clinical diagnosis of rickets was made in 35 per cent of the breast fed infants and 85 per cent of the artificially fed infants. The roentgenologists reported rickets in 35 per cent of the breast fed infants and 90 per cent of the bottle fed infants. However, the calcium-phosphorus product was constantly above 50 in the bottle fed infants. The results in nutrition and growth obtained in feeding fresh cow's milk and formulas of evaporated milk were almost identical. Evaporated milk caused fewer gastro-intestinal upsets and was less difficult to feed. It was believed that the addition of lactic acid rendered the milk more digestible and increased the level to which sugar might be added with safety to the formula.

LEMASTER, Evanston, Ill.

ON THE MOTION OF GROWTH: XVI. CLINICAL ASPECTS OF HUMAN GROWTH AND METABOLISM WITH SPECIAL REFERENCE TO INFANCY AND PRE-SCHOOL LIFE. NORMAN C. WETZEL, J. Pediat. **4**:465 (April) 1934.

Problems of growth and metabolism during infancy and early childhood are discussed. Periods of excessive gain during infancy are followed by periods of delay in growth.

Growth, gain and basal heat production are all at low tide during preschool life. Anorexia in the preschool child may be due to a relatively low demand for energy at this time of life.

LEMASTER, Evanston, Ill.

RICE POLISHINGS AS A SOURCE OF VITAMIN B COMPLEX IN INFANT FEEDING. MARTIN F. GAYNOR and ROGER H. DENNETT, J. Pediat. **4**:507 (April) 1934.

A water extract of rice polishings was added to the formulas of dried milk fed to one hundred infants. These infants gained weight more rapidly than control groups, showed no anorexia or gastro-intestinal disturbances and exhibited greater resistance to infections. The general behavior and appearance of the infants receiving vitamin B from rice polishings were far superior to those of the other infants. The authors consider it highly desirable to supplement the infant's feedings with vitamin B.

LEMASTER, Evanston, Ill.

EVAPORATED MILK AND BETA LACTOSE IN INFANT FEEDING. SIMON SKOLE, M. J. & Rec. **139**:87 (Jan. 17) 1934.

One hundred and eight selected infants were chosen for this study. They were divided into two groups: half were put on a diet of whole cow's milk and some

form of carbohydrate, and the other half were put on a diet of evaporated milk and beta lactose, which proved to be a safe, suitable and satisfactory food for the average normal infant. The evaporated milk was well tolerated by all the infants, and they showed an increase of 8/10 ounce (22.7 Gm.) more per week than the control group. The formula is uniform in composition. The fat and protein are more easily digested than the fat and protein in cow's milk. No case of clinical rickets was observed; the infants were not constipated and showed gastro-intestinal disturbances less frequently than the control group.

WILLIAMSON, New Orleans.

THE EFFECTS OF MODERATE OVERDOSAGE OF VITAMIN D AND OF VITAMINS A AND D ON THE GROWTH RATE AND LONGEVITY OF THE WHITE MOUSE. T. B. ROBERTSON, H. R. MARSTON, M. C. DAWBARN, J. W. WALTERS and J. D. O. WILSON, Australian J. Exper. Biol. & M. Sc. **9**:203, 1932; **11**:49 (March) 1933.

The rate of growth of the animals which received a moderate overdosage of vitamin D (0.05 cc. of cod liver oil concentrate) was greater between 13 and 72 weeks than that of the controls, but longevity was not affected. Fifty rat units per day of vitamin D in the form of irradiated ergosterol caused a similar increase in the rate of growth. However, this beneficial effect was not evidenced by the animals which received a considerable daily overdosage of vitamin A (460 rat units) superimposed on the 50 rat units of vitamin D. From this latter fact, it would seem that an overdosage of vitamin A antagonizes the growth-promoting effect of a moderate overdosage of vitamin D.

Both the rate of growth and the duration of life of the animals which received 50 rat units of vitamin D in the form of irradiated ergosterol daily were similar to those of the animals which received 0.05 cc. of cod liver oil concentrate daily. When, however, an excess of vitamin A (460 rat units) was simultaneously administered with the overdosage of vitamin D (50 rat units in the form of ergosterol), the expectation of life of young animals was reduced; that of the animals which survived to 750 days was not significantly altered.

GONCE, Madison, Wis.

THE SIGNIFICANCE OF VITAMINS IN PRACTICAL EXPERIENCE. LESLIE J. HARRIS, Brit. M. J. **2**:367 (Aug. 26) 1933.

The prevalence of rickets in England is discussed, as is the incidence of rickets with and without treatment. This is followed by a brief discussion of the merits of different prophylactic methods and a discussion of doses of viosterol, giving the views of various investigators as to the dose of cod liver oil alone, cod liver oil with viosterol and viosterol alone. Irradiated milk and heliotherapy as prophylactics for rickets come in for more liberal discussion. Conclusions regarding the curative treatment of rickets are derived largely from the observations of a number of well known physicians, and a word of caution against hypervitaminosis is introduced, with a brief description of the clinical manifestations. A brief outline of the action of the vitamins follows, with statements as to the value, particularly in prophylaxis, of the different vitamins. The article closes with warnings and conclusions, particular stress being laid on the fact that man cannot select his own diet intelligently.

ROYSTER, University, Va.

SOME OBSERVATIONS ON RICKETS, WITH SPECIAL REFERENCE TO ITS OCCURRENCE IN PIGS. JOHN POOLE MCGOWAN, Brit. M. J. **2**:599 (Sept. 30) 1933.

The pig apparently reacts both to a deficiency diet, especially a diet deficient in calcium, and to prophylaxis with cod liver oil and vitamin D, certainly as regards osteoporosis, in a manner far different from that of human beings. The discussion is interesting. The biochemical problem is especially stressed.

ROYSTER, University, Va.

THE USE AND MISUSE OF CARBOHYDRATES. J. H. P. PATON, Tr. Med.-Chir. Soc. Edinburgh, 1933, p. 177; in Edinburgh M. J., Oct., 1933. JOHN EASON, Tr. Med.-Chir. Soc. Edinburgh, p. 191, 1933; in Edinburgh M. J., Nov., 1933.

These two articles concerning the effects of the carbohydrate intake in Scotland may be said to be a continuation of the paper published about one year previously by Paton entitled "National Consumption of Carbohydrates—Relation to Disease." Paton, in his more recent article, suggests that improvement in the quality and reduction in the quantity of the carbohydrate intake in Scotland would produce beneficial effects on the public health.

Eaton, in his portion of the paper, is more conservative and feels that the diet should be well balanced, which may require a reduction of the sugar in some cases, although a drastic restriction of sugar may occasionally be unwise. Those interested should read the original articles and the discussion thereafter.

NEFF, Kansas City, Mo.

NEW VIEWS REGARDING THE ALIMENTARY DISTURBANCES OF NURSLINGS. H. FINKELSTEIN, Méd. inf. **40**:1 (Jan.) 1933.

The old conception that diarrhea results from direct stimulation by the lower volatile fatty acids is no longer held. Diarrhea is explained by the fact that each intestinal loop has a chyme of definite composition adapted only for that segment. Any change in composition causes the chyme to act chemically, stimulating peristalsis and producing diarrhea. This definite and specific composition is maintained by the harmonious and cooperative activities of secretion, digestion, absorption and motility. Anything that disturbs any of these processes causes a change in the chyme, with resultant diarrhea. Such disturbances can be brought about by drugs, parenteral infections, heat and an unbalanced diet.

Bacterial infection is not essential for the production of diarrhea, as a sterile chyme can cause it.

As to treatment, the author states that foods which are acid or which contain casein in the finest subdivision are the best.

Diarrhea of the large intestine is probably caused by colitis.

The author believes that toxicosis is due essentially to a lack of water in the portal system and in the liver, bringing about abnormal products in the circulation which produce an acute desiccation of the tissues. These products have been identified as belonging to the histamine and histaminiform group. The progressive exsiccosis finally prepares the foundation for a general alteration which involves all intermediate processes, the clinical termination of which is a toxic coma.

Similar pictures can be produced without an insufficient supply of water or without loss of water by way of the gastro-intestinal tract, as in parenteral infections. It can be supposed that infections injure the hepatic functions in the same manner as a lack of water, or else that the bacterial decomposition of the cellular elements produces the same capillary toxins which in exsiccosis are produced by the liver.

COPPOLINO, Philadelphia.

THE RECENT DISCOVERIES ABOUT RICKETS AND THEIR INTERPRETATION. A. B. MARFAN, Méd. inf. **40**:56 (Feb.) 1933.

Marfan states that from recent discoveries the following can be deduced with certainty: (1) that rats subjected to a diet poor in phosphorus present a dystrophy of the bones similar to that of rickets; (2) that the ultraviolet rays modify, if not rickets, at least the bone lesions it causes, and (3) that viosterol has an action analogous to that of the ultraviolet rays.

He believes that the American conception that the rachitic bone lesion is simply an insufficient calcification or a decalcification is incorrect because his histologic

researches have shown that the principal lesions consist in an irritation of the bone marrow with formation of the spongy tissue characteristic of rickets.

He states that prolonged infections and intoxications, such as syphilis, tuberculosis, bronchopneumonia, cutaneous infections and chronic nasal diphtheria, in addition to the aforementioned factors, can cause rickets.

He believes that light therapy affects only the bone lesions and has no effect on the other signs of rickets, viz., hyperplasia of lymphoid organs, anemia and muscular hypertonia.

He states that in addition to the inactive ergosterol present in the organism there is formed in the spleen in the natural state a sterol with antirachitic properties similar to viosterol. The latter affects chiefly the bone lesions; its effect on the other signs is inconstant and incomplete.

COPPOLINO, Philadelphia.

PROGNOSTIC VALUE OF STUDIES OF THE ACID-BASE BALANCE. L. RIBADEAU-DUMAS ET AL, Nourrisson **22**:65 (March) 1934.

Studies were made to determine the effect of injections of isotonic solutions of sodium bicarbonate on the alkaline reserve of the blood plasma and on the ratio between chlorides in the red blood cells and the plasma. In normal infants or in infants suffering from minor disturbances no significant variations were found in samples taken either two or twenty-four hours following the injections. In another series of infants, who were made febrile by injections of a vaccine, it was found that those who were hydrolabile and showed marked loss of weight also showed an elevation in chlorides; of the four infants showing this reaction, three subsequently died. The authors believe that studies of the chloride ratios in acutely ill infants or in those rendered febrile give valuable prognostic data.

MCCLELLAND, Cleveland.

RECURRING CRANIOTABES. JUAN P. GARRAHAN and JUAN C. TRAVERSARO, Arch. argent. de pediat. **4**:869 (Dec.) 1933.

The authors report this condition in two infants, one aged 6 months and one aged 3 months. The condition cleared up with the use of ergosterol; it reappeared again on discontinuance of the remedy and disappeared when treatment was resumed. In some cases craniotabes seems to be resistant to all forms of treatment. The authors would class these cases with cases of osteoporosis and, in later life, with cases of rachitis tarda.

SCHLUTZ, Chicago.

WET NURSING. T. VALLEDOR, Bol. Soc. cubana de pediat. **6**:33 (Jan.) 1934.

The author concludes that wet nursing has more disadvantages than advantages. It always has a limited application. There is always the danger of syphilis, both to the infant from the wet nurse and to the wet nurse from a congenitally syphilitic infant.

The wet nurse's own infant has the first right to her breast milk, at least for the first 5 months of life.

Wet nursing is not widely practiced in Cuba.

SCHLUTZ, Chicago.

TREATMENT OF ATHREPSIA. JUAN L. MORALES GONZALEZ, Med. de los niños **34**:198 (July) 1933.

The complexity of the problem of the treatment of athrepsia is in some measure due to the fact that the causes are so various; among the causes are hereditary syphilis, avitaminosis, lowered digestive tolerance in general, tuberculosis, putrefaction and intoxication, and congenital intolerance of fat, carbohydrate or albumin. In each case the cause should be determined in order to apply therapeutic measures intelligently.

The importance of breast milk is emphasized. Insulin in doses of 1 unit per day per kilogram of body weight is useful in those infants showing an intolerance to carbohydrates. Those with hereditary syphilis receive progressively increasing doses of sulpharsphenamine every four days. Thyroid extract is of value in athreptic infants to stimulate metabolism, a form of therapy first employed by Simpson in 1910 in England.

When the athreptic state is complicated by infections such as pyodermia, pyelonephritis and multiple abscesses, protein therapy has been used advantageously. Finally, the use of blood transfusions as well as the well recognized supportive measures, such as the use of Ringer's solution, physiologic solution of sodium chloride and therapy with ultraviolet rays from the mercury vapor arc in quartz, all have an important place in the management of athrepsia.

DWYER, Kansas City, Mo.

ACETONEMIC CONVULSIONS IN CHILDHOOD; SIGNIFICANCE OF THE HYPOGLYCEMIA. G. FANCONI, Jahrb. f. Kinderh. **142**:1 (Feb.) 1934.

The author reports on seven cases of convulsions in children with acetonuria. The acetonuria persisted for several days, during which dextrose tolerance tests revealed a diminished rise of blood sugar, a normal *Staubeffect* and an abnormally marked rise after the injection of epinephrine. Fanconi ascribes the disorder to transient hyperinsulinism and suggests as treatment the use of dextrose by mouth, by rectum or by injection and a diet rich in carbohydrate and low in protein and fat.

CLAUSEN, Rochester, N. Y.

FAILURE OF CAROTENE? THERAPEUTIC ACTION OF WHOLE MILK AND COD LIVER OIL IN VITAMIN A DEFICIENCY. E. WIELAND, München. med. Wchnschr. **80**:1389 (Sept. 8) 1933.

Xerophthalmia and keratomalacia, the chief manifestations of vitamin A deficiency, are rarely seen in Switzerland. They are observed more frequently in dystrophic infants during severe gastro-intestinal disturbances. Wieland reports the case of a nursling who showed dyspepsia when breast feeding was stopped at 14 days of age. The condition became worse when whole breast milk was again started, so the infant was given skimmed breast milk. The infant became atrophic, and hemorrhages, xerophthalmia and keratomalacia developed.

The diet was then changed to whole breast milk with 30 drops of a concentrated solution of carotene. The hemorrhages stopped, but the eyes became worse. The hemoglobin was 40 per cent; erythrocytes numbered 2,400,000 and platelets 200,000. This condition was considered as characteristic thrombocytopenia due to vitamin A deficiency. The infant was then given a feeding of whole breast milk with 20 Gm. of cream daily. Twenty-five drops of cod liver oil was given daily. Following this treatment the weight increased; the photophobia improved, and the discharge from the eyes stopped. After three months the infant was well enough to permit of discharge from the hospital, but he still had amaurosis.

Wieland discusses other cases of keratomalacia in infants who were fed skimmed milk. Carotene has been effective in curing vitamin A deficiency in rats, but failed to do so in his infant. The author advises the use of cod liver oil in such cases and thinks that the infant's sight might have been saved if it had been given earlier.

BRAHDY, Mount Vernon, N. Y.

NUTRITIVE VALUE OF SPERM-WHALE OIL AND FINBACK-WHALE OIL. YOSHIKAZU SAHASHI, Scientific Papers, Inst. Phys. Chem. Research (Tokyo) **20**:245, 1933.

Sperm-whale oils produced seborrhea and retarded growth in rats. Pure waxes did not do this. Fatty alcohols were toxic but did not cause seborrhea. Since waxes are not toxic, they are probably not assimilated. Seborrhea was prevented by the addition of linoleic acid and yeast.

KUGELMASS, New York.

CHANGES IN THE BLOOD PICTURE IN VITAMIN A DEFICIENCY. T. HART DE RUYTER, Nederl. tijdschr. v. geneesk. **77**:2672 (June 10) 1933.

In the course of vitamin A deficiency, rats which were not infected with Bartonella did not show any anemia of significance. Nonsplenectomized rats which were latently infected with Bartonella showed fatal anemia of the hyperchromatic type. For five months these animals had been fed a diet free from vitamin A, with the periodic addition of small quantities of this vitamin during the last eight weeks, so that they really suffered from prolonged hypovitaminosis. Bartonellae were found in the blood. This type of anemia, which probably arises from the activity of Bartonellae, is thought to be concerned with the atrophy of the whole lymphatic system which appears in the course of prolonged vitamin A deficiency.

The thymus and spleen were greatly decreased in size. The lymphopenia of the blood, which was pronounced in most instances, was also seen as an expression of the atrophy mentioned. In some animals the number of polymorphonuclear leukocytes was increased relatively and absolutely, this increase being in proportion to the extent of the infection; in others, however, leukopenia was present. Infection with Bartonella in the course of vitamin A deficiency shows the influence of this deficiency on the red and white blood cells.

VAN CREVELD, Amsterdam, the Netherlands.

## Prenatal Conditions

POSSIBLE ELECTROCUTION OF A FETUS IN UTERO. V. CATHALA, Bull. Soc. d'obst. et de gynéc. **22**:807 (Dec.) 1933.

A woman 47 years of age received a severe electric shock from a defective electric light socket during the seventh month of pregnancy. No lesion occurred at the site of injury. At eight months a living female infant with peculiar cutaneous lesions was delivered. The lesions were scablike and occurred on prominent projecting portions of the body, such as the nose, ears and knees. The skin elsewhere was normal. The scabs seemed to be somewhat old, for in some areas scar formation was taking place. The infant died when 3 days old. No Wassermann test was carried out, but microscopic evidence of syphilis could not be seen in the liver, skin or other tissues. The mother had previously given birth to nine normal infants. The author believes that the cutaneous lesions presented by the last infant were the result of the electric shock suffered by the mother. However, he mentions cases of pregnant women who received electric shocks and gave birth to infants without such lesions. The author also subjected pregnant guinea-pigs to sublethal electric shocks, and they had normal litters. However, in guinea-pigs the conditions are not the same as in man, because of the hair covering the young and the small amount of amniotic fluid. Cathala reported this case to stimulate interest in this unusual subject.

GLASER, Rochester, N. Y.

ROENTGENOGRAPHIC DIAGNOSIS OF FETAL ASCITES IN UTERO. P. BRAULT and A. TISON, Bull. Soc. d'obst. et de gynéc. **23**:36 (Jan.) 1934.

A woman who had previously delivered a normal child, 5 years of age at the time of examination, and who had aborted for unknown reasons during her second pregnancy, was brought to the hospital during her third pregnancy because of suspected hydramnion. Serologic examination of the patient and her husband showed no syphilis. A roentgenogram of the fetus was taken because of a suspected malformation, presumably cranial. The skull, however, appeared normal. The attitude of the fetus was striking. The legs were extended and spread apart, suggesting a floating frog. The superior outline of the thorax was normal, sharply widening out in its lower portion into a voluminous shadow in the region of the abdomen, because of which the diagnosis of fetal ascites was made. Several days later a fetus was expelled which presented marked ascites. The authors believe

that the attitude of the lower extremities of the fetus, due to the large size of the abdomen, is a valuable sign in the roentgenographic diagnosis of this condition in utero.

GLASER, Rochester, N. Y.

A RENAL ANOMALY. E. CARDEMIL, Arch. de hosp. d. niños, Roberto del Rio **4**: 79 (June) 1933.

This congenital anomaly consisted of a double kidney with infected hydronephrosis of the pelvis and hydro-ureter of the corresponding side.

SANFORD, Chicago.

CONGENITAL AORTIC STENOSIS COMBINED WITH ENDOCARDIAL HYPERPLASIA IN THE NEW-BORN. E. DISSMAN, Frankfurt. Ztschr. f. Path. **43**:476, 1932.

A case is reported. The aortic valve in the region of the bulbus revealed three areas of thickening parallel to the aorta. One of the areas formed a ball-like structure measuring about 4 mm. in diameter, which almost completely obstructed the lumen of the aorta. The endocardium of the left ventricle corresponding to the interventricular septum just beneath the aortic valve was white and measured as much as 2 mm. in thickness. Its surface was smooth in some portions and rough in others, resembling the trabeculation of the apex of the left ventricle. Dissman believes that the thickenings of the endocardium and in the region of the aortic valve were the result of primary hyperplasia of the embryonal texture of the valvular apparatus and of the endocardium. He does not believe that an intra-uterine inflammation was responsible for these changes.

O. SAPHIR, Chicago. [ARCH. PATH.]

PECULIAR MALFORMATION OF THE PULMONARY VEINS. H. REBENSBURG, Frankfurt. Ztschr. f. Path. **44**:137, 1932.

A male infant died suddenly about three months after birth. The left ventricle was much smaller than the right. The aorta showed no changes, and the ductus arteriosus was obliterated. There was no communication between the left auricle and the lungs, the pulmonary veins not being in their normal location. The foramen ovale was wide open and round, measuring 5 mm. in diameter, and the right auricle was three times as large as the left. Into the right auricle opened the coronary veins and the superior and inferior venae cavae, in addition to another vein which extended from the right upper lobe of the lung into the auricle. Another pulmonary vein took its course from the lower lobe of the right lung to the lower lobe of the left lung, forming a bridge between these lobes. This vein also extended throughout the entire left lung to the region of the hilus, collecting smaller tributaries, and opened into the superior vena cava just above the right auricle. The pulmonary arteries were normal. The arterial blood took the following courses from the lung to the heart: from the right upper lobe into the right auricle; from the right lower lobe to the left lung and from there to the superior vena cava and also into the right auricle; from the right auricle through the patent foramen ovale into the left auricle.

O. SAPHIR, Chicago. [ARCH. PATH.]

CONGENITAL ADENOMYOSIS. R. WEYENETH, Frankfurt. Ztschr. f. Path. **46**:43, 1933.

In forty-two girls aged up to 14 years four instances of endometrial structures within the myometrium were found. Various other congenital anomalies were present also, including true hermaphrodism, a case of which is described in detail. The author also describes a case of uterus didelphys with endometrial structures throughout the entire left half. Adenomyosis of the uterus and a similar condition

of the tube should be regarded as malformations resulting from extension of the endometrium into the myometrium during the fetal period or from cutting off of epithelial elements of the mullerian duct.

O. SAPHIR, Chicago. [ARCH. PATH.]

SOME UNUSUAL MALFORMATIONS. F. GRÖGLER, Virchows Arch. f. path. Anat. **289**:430, 1933.

Four fetuses with craniorachischisis, two acardiac monstrosities and one phocomelus with the defect limited to one upper extremity are described. Craniorachischisis is ascribed by Grögler to the action of external factors. The phocomelus was a woman who died at the age of 21 of pulmonary tuberculosis. The forearm, two phalanges and part of the carpus were absent.

SCHULTZ, Evanston, Ill. [ARCH. PATH.]

A CASE OF CLEIDOCRANIAL DYSOSTOSIS. P. LADEWIG, Virchows Arch. f. path. Anat. **291**:542, 1933.

The rare condition that P. Marie and Sainton described in 1898 under the name "dysostosis cleidocranialis hereditaria" is characterized by small body stature; maldevelopment of the cranial vault, with prominence of the frontal and parietal regions; late or deficient ossification of sutures and fontanels, which are closed by multiple irregular masses and nodules of bone; an abnormally small face; deformity of the jaws with many unerupted teeth, and bilateral partial or complete absence of the clavicles. There may be other skeletal deformities. Anatomic descriptions are meager. This Ladewig ascribes to the fact that the persons with the deformities are usually well psychically and as regards their general health; the condition goes unrecognized and escapes the pathologist. In the case described by Ladewig, that of a woman who died at the age of 60 years of carcinoma of the uterus, the body had gone first to the anatomic institute, where it had been partially dissected before its peculiarities were recognized. His description is therefore limited chiefly to the skull. He concludes that the condition is a congenital and probably hereditary one, characterized by diminished capacity of expansion on the part of the ossification centers, especially those of the membranous bones. Similar but less extremely diminished growth energy is evident in the rest of the skeleton.

SCHULTZ, Evanston, Ill. [ARCH. PATH.]

FETAL PERITONITIS DUE TO APPENDICITIS. R. KNEPPER, Virchows Arch. f. path. Anat. **291**:741, 1933.

A child who died at the age of 2½ months began to lose weight rapidly at the eighth week of life, had diarrhea and fever, became dehydrated and died. Necropsy revealed the presence of organized fibrous peritoneal adhesions, with an encapsulated mass of exudate in the ileocecal region. The distal end of the appendix was open and was embedded in the mass of exudate. The latter was partly calcified and contained meconium.

SCHULTZ, Evanston, Ill. [ARCH. PATH.]

BIRTH AND PATHOLOGIC-ANATOMIC PECULIARITIES IN A RARE MONSTER. M. C. BOON VAN OCHSSÉE, Nederl. tijdschr. v. geneesk. **78**:1131, March 10, 1934.

This is a description of the pathologic-anatomic features of a monster born with artificial aid, the second child of mono-ovular twins. The other child was normal.

VAN CREVELD, Amsterdam, The Netherlands.

## Diseases of New-Born

MULTIPLE ULCERS OF THE STOMACH IN A NEWBORN INFANT WITH STAPHYLOCOCCUS SEPTICEMIA. ETHEL C. DUNHAM AND MERRIWELL T. SHELTON, J. Pediat. **4**:39 (Jan.) 1934.

Many ulcers were found in the stomach of a new-born infant who died of staphylococcic septicemia of umbilical origin. Histologic sections of the wall of the stomach showed no cellular reaction around the bases of the ulcers, but there were thrombi in the blood vessels. There were also numerous thrombi in the pulmonary arterioles. The gastric and pulmonary lesions were apparently embolic in origin.

LEMASTER, Evanston, Ill.

RUPTURE OF THE STOMACH IN NEWBORN INFANTS. ETHEL C. DUNHAM and RICHARD M. GOLDSTEIN, J. Pediat. **4**:44 (Jan.) 1934.

Two cases of rupture of the stomach in new-born infants are reported. Only nine similar cases have been described. In the authors' cases both children showed vomiting, abdominal distention and positive blood cultures for Bacillus coli during life. The cause of rupture of the stomach could not be definitely established in either case, but several theories are discussed. It is suggested that when this condition is suspected a roentgenogram of the abdomen should be taken to reveal the presence or absence of free air in the peritoneal cavity.

LEMASTER, Evanston, Ill.

TREATMENT OF INTRACRANIAL HEMORRHAGE IN THE NEWBORN. MARK HORSTEIN, M. J. & Rec. **138**:162 (Sept. 6) 1933.

The symptoms and prophylaxis of this condition are first described. Treatment consists of the insertion of a trocar and cannula into the compressing mass after an accurate estimation has been made of the approximate location. The trocar is then withdrawn, and a Luer syringe is attached to the cannula, which is used for aspiration. The treatment is fully discussed. Six cases in which this method was used are reported.

WILLIAMSON, New Orleans.

SOME PROBLEMS OF THE NEWBORN THE FIRST FEW DAYS OF LIFE. HERMAN JAHR, Nebraska M. J. **19**:86 (March) 1934.

This article is based on nine years' experience in the service for the new-born of the University of Nebraska College of Medicine. Mild cases of clubfoot have frequently been corrected by the use of adhesive strips. Severe cases need special orthopedic procedures. Supernumerary toes or fingers are removed early by surgical procedure. Tonguetie has been infrequent; only twice in the four years has it been necessary to clip the frenum. Circumcision is performed early, when indicated by a tight, adherent prepuce. It is needed infrequently. Inanition fever and loss of weight are prevented and treated by the administration of 2 ounces (59 cc.) of 17 per cent sugar every four hours, beginning six hours after birth. Reactions to silver should be differentiated from gonorrheal infection of the eyes by frequent microscopic examination. Engorgement of the breasts, if let alone, causes no trouble. In regard to cerebral hemorrhage, infants with massive hemorrhage either are born dead or die soon after birth. In infants with moderate hemorrhage, who are too often left with either a physical or mental deformity, 10 cc. of whole blood is injected twice daily as long as there is evidence of bleeding, and spinal puncture is carried out two or three times daily. In mild hemorrhages with only vague symptoms the same therapeutic measures are used. In asphyxia neonatorum carbon dioxide with oxygen is used. In the premature infant, nutrition is maintained by frequent concentrated small amounts of food; i. e., 1 or 2 drachms (3.8 or 7.6 cc.) is given every hour for the first few days. Breast milk is best, and evaporated milk with lactic acid is second best. The maintenance of heat is important, and quiet is essential.

HAMILTON, Omaha.

ASPHYXIA NEONATORUM. FRANKLIN G. RILEY, New Orleans M. & S. J. **86**:301 (Nov.) 1933.

Since he is proud of his state's (Mississipi) record of low infant mortality and since the statistics show that the mortality would be still lower if deaths occurring on the first day could be prevented, the author urges cooperation between obstetricians and pediatricians in the matter of asphyxia neonatorum. After describing the etiology and biochemical reactions and the prophylactic treatment for the mother in asphyxia neonatorum, the author gives details of treatment in deliveries in both the home and the hospital, emphasizing the pediatrician's duty in assisting the obstetrician in resuscitation.

SMITH-FINE, Dallas, Texas.

THE CHEMICAL CONSTITUTION OF THE BLOOD IN NEW-BORN BABIES AND SUCKLINGS. G. DE TONI, Arch. ital. di pediat. e puericult. **2**:1 (Sept.) 1933.

This is a monumental work, more than one hundred pages long, including more than four hundred references to the literature. The author has attempted to consolidate the physiologic foundations of blood chemistry in children of this age group as a basis for the complete study of the pathologic blood chemistry. He presents complete tables of values for nonprotein nitrogen, urea, uric acid, preformed and total creatinine, lecithin, dextrose, chlorides, calcium, magnesium, sodium, potassium and inorganic phosphorus. These tables are compiled from the results of his own researches as well as the published results of other workers and are easily intelligible, even to one who does not read Italian.

DOOLEY, Kent, Conn.

ABANDONMENT OF THE SWINGING METHOD OF RESUSCITATION OF SCHULTZE. H. CRAMER, München. med. Wchnschr. **80**:1546 (Oct. 6) 1933.

Formerly, the Schultze technic of resuscitation was a firmly established dogma. This method was practically the only treatment for asphyxia mentioned in the old textbooks. The importance of reestablishing the normal respiratory stimulus was overlooked. Cramer thinks that for this purpose lobeline is indispensable.

That the Schultze method frequently causes or aggravates cerebral injuries, tears in the dura and subdural hemorrhages was first emphasized by pediatricians. The method may also cause aspiration of the amniotic fluid and cervical mucus. These can be removed most satisfactorily by suction with a tracheal catheter.

BRAHDY, Mount Vernon, N. Y.

THE PANCREATIC ISLETS OF THE NEW-BORN INFANT OF A DIABETIC MOTHER. K. A. HEIBERG, Virchows Arch. f. path. Anat. **287**:629, 1933.

A woman with severe diabetes mellitus passed through pregnancy and delivery without any untoward events. The diabetic condition improved after delivery. The infant died two days after birth with symptoms of hypoglycemia; the blood sugar was 60 mg. per hundred cubic centimeters. Microscopic examination of the pancreas revealed marked hypertrophy and hyperplasia of the islets.

W. SAPHIR. [ARCH. PATH.]

HYDROPS UNIVERSALIS FOETUS ET PLACENTAE AND ICTERUS GRAVIS NEONATORUM. J. C. BEKER and J. H. SMIDT VAN GELDER, Nederl. tijdschr. v. geneesk. **77**:1545 (April 8) 1933.

The history and treatment of a child suffering from icterus gravis are described. The mother had given birth to two healthy children, and after that to two stillborn children with icterus gravis.

The explanation is given that the disease is caused by oxygen deficiency early during fetal life, as modern investigations pointed out in discussing the origin of icterus neonatorum.

In hydrops and icterus gravis marked reaction of the blood-preparing organs to marked oxygen deficiency is to be accepted. The treatment advised is giving iron and liver to the mother during pregnancy and decreasing the quantity of fetal blood by letting blood from the cord directly after birth and replacing it with transfused blood.

VAN CREVELD, Amsterdam, the Netherlands.

## Acute Infections

NEUTRALIZATION OF POLIOMYELITIS VIRUS BY THE SERUM OF NORMAL ADULTS OF NASHVILLE, TENN. N. PAUL HUDSON and W. LITTERER, J. Infect. Dis. **53**:304 (Nov.-Dec.) 1933.

Hudson and Litterer found that twenty-one of twenty-five specimens of undiluted serum taken from twenty-five normal white residents of Nashville, contained virucidal antibodies. Sixteen of the twenty-one specimens were potent in dilutions up to 1:5.

TOOMEY, Cleveland.

EXPERIMENTAL PERTUSSIS. H. MACDONALD and E. J. MACDONALD, J. Infect. Dis. **53**:328 (Nov.-Dec.) 1933.

The authors were able to produce whooping cough in boys by instilling suspensions of fresh cultures of Bacillus pertussis, but they could not produce the disease by instilling filtrates of the fresh cultures. They believe that this finding rules out the rôle of a filtrable virus in the etiology of the disease.

TOOMEY, Cleveland.

TETANUS TOXOID IN PROPHYLAXIS AGAINST TETANUS. DAVID H. BERGEY and SAMUEL ETRIS, J. Infect. Dis. **53**:331 (Nov.-Dec.) 1933.

Bergey and Etris showed that injections of tetanus toxoids into guinea-pigs brought about a high degree of protection against the effect of injections of tetanus bacilli. They believe that human beings can also be protected, and that the use of tetanus toxoid would prevent the hypersensitization to horse serum which sometimes occurs from passive immunization with tetanus antitoxin.

TOOMEY, Cleveland.

THE PURIFICATION OF DIPHTHERIA TOXOID. GEORGE F. LEONARD and AUGUST HOLM, J. Infect. Dis. **53**:376 (Nov.-Dec.) 1933.

None of the methods used by Leonard and Holm have fulfilled all the requirements which they have set up as necessary for the purification of toxoid on a large scale. Precipitation by ammonium sulphate with subsequent dialysis, precipitation by acetone and that by alum gave the highest yields of toxoid as measured in terms of flocculation with antitoxin. Toxoids precipitated by alum are superior in immunizing value to crude toxoids.

TOOMEY, Cleveland.

TREATMENT OF DIPHTHERITIC PARALYSIS WITH ANATOXIN. A. CUSATELLI, Bull. Soc. de pédiat. de Paris **30**:128 (Feb. 16) 1932.

A girl, 15 years old, had a sore throat from which diphtheria bacilli were recovered about ten days later, at which time paralysis of the soft palate began to develop. More than three weeks later she showed complete paralysis of the soft palate. At this time a test made with anatoxin (Ramon) was positive. She was given 0.25 cc. of anatoxin, and three more injections of 0.5, 1 and 1.5 cc., respectively, at intervals of two days. After the second dose she showed marked improvement, and recovered completely after the third dose.

BENJAMIN, Montreal, Canada.

ACUTE ETHMOIDITIS WITH CELLULITIS IN THE COURSE OF SCARLET FEVER; INTERVENTION; CURE. BABONNEIX, HALPHEN and WILM, Bull. Soc. de pédiat. de Paris **30**:583 (Nov. 15) 1932.

A boy, 5 years old, with scarlet fever, had a swelling on the left side of the bridge of the nose, edema of the left eyelids and exophthalmos of the left eye. The nasopharyngeal cavity had been the seat of infection for a long time. Roentgenologic examination revealed cloudiness of the left ethmoid and maxillary sinuses. These were explored surgically; the mucous membrane lining them was inflamed, and there was cellulitis of the surrounding tissues; no frank pus was located. The child made an uninterrupted recovery and left the hospital a month after the operation.

BENJAMIN, Montreal, Canada.

PREVENTION AND ATTENUATION OF MEASLES BY THE USE OF SERUM IN AN INFANT SERVICE. P. LEREBOULLET and J. VANIER, Bull. Soc. de pédiat. de Paris **30**:585 (Nov. 15) 1932.

A number of infants from 6 to 18 months of age were given convalescent measles serum. Most of the infants received a dose of 3 cc.; a few were given a dose of 5 cc. A large majority of those exposed did not develop measles, while a few had a mild attack.

BENJAMIN, Montreal, Canada.

INFANTILE PARALYSIS WITH RAPID CURE. BABONNEIX and WILM, Bull. Soc. de pédiat. de Paris **30**:639 (Dec. 20) 1932.

A girl, aged 14, had poliomyelitis with extensive paralysis of all the extremities. She was given antipoliomyelitic serum intramuscularly and intrathecally after the onset of paralysis, which was present on her admission to the hospital. She was further treated with repeated roentgenologic irradiations of the spinal column. Ten days after the onset of the paralysis the condition was markedly improved, and a little over a month after her admission, which corresponded to the onset of the paralysis, she was discharged as normal.

BENJAMIN, Montreal, Canada.

THE DIAGNOSTIC VALUE OF MANDELBAUM'S REACTION IN TYPHOID. IGNAZIO GATTO, Pediatria **41**:726 (June 1) 1933.

Gatto reports on Mandelbaum's reaction in the blood serum of (1) normal children, (2) patients with typhoid fever and (3) children immunized with typhoid vaccine. He found the reaction specific for typhoid fever, but not present in all cases; at times it reacts independently of the blood serum agglutination. The reaction is not uniformly met with in the blood of children vaccinated with typhoid vaccine.

SIGNORELLI, New Orleans.

CLINICAL AND ELECTROCARDIOGRAPHIC STUDIES OF DIPHTHERIA AND THE CARDIOVASCULAR APPARATUS. WILLY SCHWARZ, Pediatria **41**:737 (June 1) 1933.

Schwarz describes in detail what is known of the pathogenesis and anatomic pathologic processes of diphtheritic intoxications and refers to the myocardial disturbances described in such cases. Basing his report on ninety-eight cases of diphtheria which he studied clinically and electrocardiographically, he refers in minute detail to each observation, and summarizes thus: Among the most frequently encountered and most important disturbances are those of conduction, with which most often there is the coexisting phenomenon of extrasystole. He explains the diagnostic and prognostic importance of electrocardiographic observations and traces the complex clinical cardiovascular manifestations met with in diphtheria.

SIGNORELLI, New Orleans.

RELATION BETWEEN SCARLET FEVER AND TUBERCULOSIS. L. PICCALUGA, Riv. di clin. pediat. **31**:1186 (Oct.) 1933.

Piccaluga found that about 80 per cent of tuberculous children contracting scarlet fever showed a negative reaction to tuberculin during the course of the disease; most of the observations were made in the stage of desquamation. This loss of allergic reaction does not signify, he thinks, a loss of immunity to tubercle bacilli, but rather local cutaneous changes in scarlet fever. Scarlet fever is strikingly different from measles in its effect on tuberculous patients; many patients trace the onset of an active tuberculous reaction to measles, but few do to scarlet fever. The author's clinical experience is that scarlet fever seldom, if ever, lights up a tuberculous focus.

HIGGINS, Boston.

DIPHTHERITIC ANGINA IN THE IMMUNIZED. JUAN CARLOS MUNYO and EUGENIA YANNUZZI, Arch. pediat. d. Uruguay **4**:167 (May) 1933.

Six cases are reported. In one the length of time elapsed since immunization was five years; in one, three years, and in four, two years. In two of the patients severe symptoms of diphtheria developed which required antitoxin in moderate dosage. The other four patients recovered without the use of serum. The authors believe that immunity in immunized persons varies in different ones and is dependent somewhat on the length of time elapsed since immunization.

SCHLUTZ, Chicago.

DIPHTHERIA IN BARCELONA. MARTINEZ VARGUS, Med. de los niños **34**:193 (July) 1933.

A long article on diphtheria running through several numbers is brought to a conclusion this month. The frequency of diphtheria in Spain and the complications of bronchopneumonia and laryngitis are noteworthy. The history of diphtheria immunization is reviewed, and mention is made of the fatalities attending the early use of toxin-antitoxin in Dallas, Texas, and in Queensland, Australia.

The technic of vaccination is described, including the hypodermic injection of 1 cc. of antitoxin, the second dose to follow in fifteen days and the third in twenty-one days. The cutaneous administration of an ointment containing diphtheria toxin-antitoxin after the method of Löwenstein is described. Löwenstein has used this inunction 18,000 times in 6,000 children in two years. After forty-five days, none of these had acquired diphtheria. The technic consists of cleansing the arm with benzine and alcohol, then applying the ointment by gentle friction for two or three minutes. After fifteen days the second inunction is given and the third fifteen days later. The immunity is complete in forty-five days.

DWYER, Kansas City, Mo.

DETAILS OF THE EPIDEMIC OF POLIOMYELITIS IN SZEGED IN 1932 AND EXPERIENCES WITH PROPHYLACTIC INJECTIONS OF BLOOD. K. WALTNER, Arch. f. Kinderh. **100**:147 (Sept. 29) 1933.

In 1932, there were two hundred cases of poliomyelitis in Szeged and vicinity. Most of the cases occurred during August, September and October. Although children under 5 years of age were chiefly affected, there were many older children who became ill. Several children were given a prophylactic injection of from 10 to 20 cc. of whole blood from the parents, administered intramuscularly. All cases of poliomyelitis developing in these protected children were very mild. The author strongly advocates the use of adult whole blood for the protection of children during an epidemic of poliomyelitis.

LEMASTER, Evanston, Ill.

MODIFICATION OF VACCINATION FOR SMALLPOX BY THERAPEUTIC MEANS. E. BARLA-SZABÓ, Arch. f. Kinderh. **101**:1 (Dec. 19) 1933.

Roentgen irradiation at the site of a reaction to vaccination on the eighth day after inoculation caused disappearance of systemic symptoms such as fever, malaise

and loss of weight, and also produced rapid drying of the pustule and decrease in the size of the inflammatory areola. There was no effect when the irradiation was applied before the pustular stage. It is claimed that this method does not lessen the general immunity to smallpox. Irradiation is recommended for patients showing an abnormally severe reaction to vaccination, for delicate children and for healthy children in whom an intercurrent disease appears at the height of the reaction.

LEMASTER, Evanston, Ill.

TITRATED ANTITOXIC SERUM IN SCARLET FEVER. S. N. ROSANOW, N. J. SKWORZOW, A. A. KISLOWSKAJA, B. S. HIRSCHBERG, A. D. SABULONOWA, J. L. SACHS and S. N. DOLBNINA, Jahrb. f. Kinderh. **138**:67 (Jan.) 1933.

In severe cases of scarlet fever large doses of serum are needed (from 20,000 to 25,000 units), and early injection is necessary. Toxic symptoms may not be relieved, especially in young children. In most of the hyperacute cases studied, tuberculosis was found. In moderately severe cases serum cut short the initial illness; the complications were fewer, and mortality was less. In these cases the dose consisted of from 12,000 to 15,000 units. In mild cases treatment with serum is not usually advisable, because of the untoward effects of serum sickness. However, patients treated with serum had fewer complications. The report is based on 595 patients treated with serum and 697 not so treated.

CLAUSEN, Rochester, N. Y.

SCARLET FEVER AND HEART FAILURE. W. J. MOLTSCHANOW, Jahrb. f. Kinderh. **138**:129 (Feb.) 1933.

The author reports seventeen cases of heart failure following endocarditis which developed during or soon after scarlet fever. In two cases the scarlet fever was subtoxic, requiring treatment with serum. In one case the endocarditis was observed in the second week and in fifteen in the period from the third to the eighth week. Two of the patients died of cardiac decompensation; at autopsy one of these showed changes typical of rheumatism. In fourteen patients polyarthritis accompanied the first signs of endocarditis.

CLAUSEN, Rochester, N. Y.

PNEUMONIA SECONDARY TO MEASLES. RICHARD GROSSER, Jahrb. f. Kinderh. **138**: 200 (Feb.) 1933.

Although pneumonia secondary to measles usually shows a mixture of various forms at autopsy, several types may be distinguished according to their clinical course: (1) croupous; (2) septic-toxic, with a negative urobilinogen reaction (Ehrlich), and in some cases abscesses; (3) septic-toxic, with a positive Ehrlich reaction, in which the prognosis is grave, and (4) localized peribronchial or perivascular infiltration. The characteristics of pneumonia secondary to measles are: (1) simultaneous or consecutive appearance of the various types, (2) frequently a relapse after several days or weeks, (3) delayed resolution and (4) a certain regularity in the duration of fatal cases.

CLAUSEN, Rochester, N. Y.

TREATMENT OF WHOOPING COUGH WITH VACCINE. ANNE REHMANN, Klin. Wchnschr. **10**:1358 (July 18) 1931.

Fifty-eight children, of whom thirty-two were under 1 year of age and twenty-four in the second year of life, were treated with injections of nonspecific protein. They had from sixteen to twenty paroxysms in twenty-four hours and frequent vomiting; bronchopneumonia occurred in three of them. In forty-seven other children vaccine was used, in some pertussis vaccine alone (containing 2,000,000,000, 4,000,000,000 6,000,000,000 and 8,000,000,000 bacilli per cubic centimeter) and in others a mixed vaccine. Injections were given intramuscularly every third day, from six to seven doses being used, beginning with 500,000,000 and increasing to

6,000,000,000. Slight febrile reactions occurred within from four to five hours, and during these reactions the blood picture showed an increase in the number of neutrophils and the appearance of immature forms. In the children treated with vaccine the average duration of the whooping cough was about five weeks, but a striking improvement was noted after the second or third injection. The paroxysms were reduced to five or six in twenty-four hours. Vomiting occurred in only three children. Appetite was improved.

In an infant station nine infants were left with a child who had whooping cough, infected by his own mother. These exposed infants were given three doses of vaccine at three day intervals beginning with 2,000,000,000 and increasing to 6,000,000,000. Manifest pertussis did not develop in any of these infants. In all of them there was a marked lessening of the sedimentation time, which confirms the report that in the catarrhal stage of pertussis there is a lowering of sedimentation time. Whether vaccine therapy has a specific effect cannot be said with certainty.

SILVERMAN, Syracuse, N. Y.

MIXED INFECTIONS WITH BACILLUS INFLUENZAE AND PNEUMOCOCCI IN SCARLET FEVER. HELENE SSAWRIMOWITSCH and W. D. ZINSERLING, Jahrb. f. Kinderh. **138**:319 (March) 1933.

Of 348 patients with scarlet fever examined at autopsy, 32 had bronchitis, 213 had pneumonia, 24 had pulmonary abscesses without pneumonia or bronchitis, and 79 had normal lungs. In 21 of the 213 cases of pneumonia the condition was lobar, in 128 lobular, in 32 peribronchial and in 32 not classified. Cultures were found to be positive as follows:

| | Number of Cases | Pneumococcus | Streptococcus | Bacillus Influenzae |
|---|---|---|---|---|
| Lobar pneumonia | 15 | 13 | 5 | 5 |
| Lobular pneumonia | 60 | 26 | 39 | 24 |
| Peribronchial pneumonia | 11 | 6 | 10 | 6 |

During coincident epidemics of scarlet fever and influenza in 1928 and 1929, the authors observed 16 patients with fibrinous membranes in the nose, throat, larynx, trachea, esophagus or stomach; cultures revealed no diphtheria bacilli but often revealed streptococci and Pfeiffer's bacillus. The pulmonary abscesses were usually due to streptococci.

CLAUSEN, Rochester, N. Y.

SPECIFIC TREATMENT OF POLIOMYELITIS. FRANZ HAMBURGER, Wien. klin. Wchnschr. **46**:295 (March 10) 1933.

Hamburger states that during the past ten years many investigators have pointed out that serum from immunized horses, as well as convalescent serum and even normal serum from adult man, exerts a favorable influence on the course of epidemic poliomyelitis. Statements in the literature regarding this fact are to be found in the book "Prophylaxe und Therapie der Infektionskrankheiten und Idiosynkrasien mit spezifischen und unspezifischen Mitteln" by Busson, who makes recommendations similar to those of Hamburger with regard to the specific treatment of poliomyelitis.

The latter suggests that the physician called to attend a patient whose condition arouses a suspicion of poliomyelitis immediately inject blood from either the father or mother or both into the child intramuscularly. The reason for this action is that it saves time, since it is well known to what an extent success in serum treatment depends on early application.

ABT, Chicago.

SERUM TREATMENT OF ACUTE INFANTILE PARALYSIS. HERBERT OREL, Wien. klin. Wchnschr. **46**:590 (May 12) 1933.

Orel states that recent epidemics of poliomyelitis have reawakened interest in the treatment of this disease. Special attention has been given to tests with

immunobiologic therapy, and a brief description of experiences in the specific prophylaxis and therapy of poliomyelitis to date is desirable.

Theoretically, two ways are open to immunobiologic combating of poliomyelitis, as in every other infectious disease, namely, active and passive.

Flexner and Lewis successfully immunized monkeys with increasing doses of the virus. In man this method is not desirable, because of the uncertainty as to dosage. Active immunization with the chemically, i. e., physically, weakened virus is still in the experimental stage, even in animals. Because of the infrequency of the disease, active immunization should never be attempted in man, especially in epidemics in which protection would be too late in developing.

Passive immunization, as demonstrated by neutralization tests, is possible with convalescent serum. Only persons 3 years or older may be used as donors of blood, and at the time of the withdrawal of blood they should show no evidences of infectious disease, notably syphilis (examination to include serologic tests) and tuberculosis (physical and clinical examination). The blood is taken under strict observance of asepsis. For this purpose several syringes of from 30 to 50 cc. capacity are used, which are equipped with a short connective piece of sterile rubber hose between the cannula and the cylinder. The blood is set out in sterile retorts or glasses, closed with sterile rubber stoppers, at room temperature; and after it has become coagulated it is stored in a cool place and sent to the institution equipped for the manufacture of the serum in the least time. The name and age of the donor, as well as the history of the course of the disease, the date of the transfusion and the amount of the blood donated, are sent to the institution.

Suggestions as to the amount of blood to be taken from an adult vary according to the authors, and the question is therefore best decided in accordance to whether blood is to be taken frequently. The Canadian authors advise taking 100 or, at the very most, 200 cc. The German authors generally advise taking more. According to reports in the literature, serum taken from one to two months after the onset of the disease should be the most effective. No studies have been made as to the durability of poliomyelitic convalescent serum. The difficulties encountered in the production of human serum have led to the search for an animal serum, as in other infectious diseases.

Testing of streptococcic serum, as advocated by Rosenow, and of common horse serum, as recommended by Rowan, are not considered, since only these authors still believe in their effectiveness.

Pettit treated sheep with spinal fluid from monkeys that died of poliomyelitis and professed to have obtained specific effective serum. He also successfully immunized monkeys, but the amount of serum obtained was small. At present the serum is obtained through immunization of horses. However, not every animal is able to produce antibodies. French authors, especially Etienne, are in favor of Pettit's serum; and Bremer (Munich) also believes in its effectiveness. The value of Pettit's serum has been proved in neutralization tests (Fairbrother). Again, others, chiefly Stewart and Haselbauer, disclaim any effectiveness. Pettit claims to have compounded a serum from three to four times as effective as that prepared from the blood of convalescent human beings.

The Department of Public Health of Vienna obtained Pettit serum from the Pasteur Institute during 1931, and this was distributed in Vienna, Linz and other smaller cities. It was applied in doses of from 10 to 20 cc. to patients of the Vienna University Children's Clinic. In only one of the ten patients so treated was the disease in the preparalytic stage, namely, a girl, who three days later experienced promptly recurring paralysis of the right eyelid. An accompanying effect of the serum which was observed aside from the serum sickness was painful swelling at the place of injection in three cases. Examination after one year disclosed two cures in the remaining nine children, the other children displaying more or less apparent paralysis, with atrophy of the muscles. In the Karolinen Kinderspital of the Vienna Community, ten children were treated with like serum. Here 10 cc. was introduced intraspinally and 30 cc. intramuscularly, a seemingly more intensive treatment. Two of the children died on the second and fifth days,

respectively, of Landry's paralysis; one was dismissed unimproved; two were improved, and five were dismissed as cured. With so small a number as twenty patients for comparison with nontreated patients, judgment on the value of Pettit serum must be withheld.

ABT, Chicago.

HERPES ZOSTER AND VARICELLA. B. P. B. PLANTENGA, Maandschr. v. kindergeneesk. **2**:403 (June) 1933.

Three infants contracted varicella in consequence of contact with the mother who suffered from herpes zoster caused by the use of arsenic.

VAN CREVELD, Amsterdam, the Netherlands.

TETANUS. C. J. VAN HOUWENINGE GRAFTDYK, Nederl. tijdschr. v. geneesk. **77**:1093 (March) 1933.

A brief review is given of the prevailing opinions about the prevention and treatment of tetanus, which appear to be unanimous as to the means of prevention but not as to treatment, about which there is a great deal of confusion.

The ways in which tetanus leads to death are discussed, and it is shown that acute heart failure by poisoning of the centers forms the greatest difficulty in treatment, as one cannot forsee or prevent this fatality. The necessity of further investigation in this direction is discussed.

VAN CREVELD, Amsterdam, the Netherlands.

PARATYPHOID FEVER, TYPE C, CAUSED BY SALMONELLA HIRSCHFELDII. J. VAN DER HOEDEN and L. A. HULST, Nederl. tijdschr. v. geneesk. **77**:1639 (April 15) 1933.

A boy, aged 14 years, suffering from paratyphoid fever, type C, was ill for six months; he complained of general ill feeling and of extensive swelling of the lymph glands in the neck, which increased notwithstanding irradiation. Salmonella Hirschfeldii was found in the new growth, tissue from which was obtained by puncture of the tumor, and in the blood, the spleen and the liquid found in the pleural cavity. In the sputum, the bile and the kidneys, however, it was constantly absent. Furthermore, the patient's serum showed a strong agglutination with S. Hirschfeldii (Widal's reaction). The tumor was found to be an endothelioma. It was impossible to determine whether the new growth resulted from the infection with Salmonella or whether the infection occurred at a later stage of the tumor.

VAN CREVELD, Amsterdam, the Netherlands.

A CASE OF TUBERCULOSIS. E. GORTER, Nederl. tijdschr. v. geneesk. **77**:1874 (April 29) 1933.

Gorter describes the clinical course of a case of pulmonary tuberculosis and gives a survey of the different methods available for judging the activity of tuberculosis.

VAN CREVELD, Amsterdam, the Netherlands.

A CASE OF WEIL'S DISEASE NOT ACQUIRED BY SWIMMING. A. W. J. H. HOITINK, Nederl. tijdschr. v. geneesk. **77**:4278 (Sept. 19) 1933.

A case of Weil's disease is reported in a boy, aged 16. The diagnosis was based on clinical signs and was corroborated later by the serologic reactions. Infection had probably occurred through a lesion in the skin of the hand while the patient was handling fish recently caught.

VAN CREVELD, Amsterdam, the Netherlands.

## Chronic Infections

THE CLINICAL VALUE OF THE PRESUMPTIVE KAHN TEST. TOMAS CAJIGAS, Ann. Int. Med. **7**:114 (July) 1933.

The author advocates the use of the presumptive Kahn test as a further check for the routine Wassermann and Kahn tests. He is of the opinion that the presumptive test is 8.2 per cent more sensitive than the Wassermann test and 5.5 per cent more sensitive than the standard Kahn test. Because of the high sensitivity of the presumptive test, a negative reaction is of more value than a similar reaction obtained by a less sensitive method. The author believes that the presumptive test is of special value in primary syphilis, latent syphilis and neurosyphilis and in determining when to discontinue treatment.

READING, Galveston, Texas.

A BRIEF REVIEW OF FEVER THERAPY IN NEUROSYPHILIS. HARRY BECKMAN, Arch. Dermat. & Syph. **28**:309 (Sept.) 1933.

This article on fever therapy in neurosyphilis is especially practical for the practicing physician. The author reviews the literature up to date. He includes the various means of producing fever, i. e., the three types of malaria, relapsing fever, rat-bite fever, typhoid-paratyphoid vaccine, injections of sulphur and the several types of diathermy. He gives specifically the method of treatment and the relative value and safety of each. Statistics show 20 per cent recovery through malarial therapy to the same social and financial status as before the neurologic involvement. However, for the more recently treated patients 30 per cent is nearer the correct value. The author believes that typhoid-paratyphoid vaccine treatment is the method of choice for the average general practitioner.

DULIN, Iowa City.

BISMUTH COMPOUNDS IN THE TREATMENT OF ACTIVE SYPHILIS; A CLINICAL AND LABORATORY STUDY OF TWO HUNDRED AND EIGHTY-SIX CASES OF CUTANEOUS SYPHILIS. S. IRGANG, E. R. ALEXANDER AND A. M. SALA, Arch. Dermat. & Syph. **28**:320 (Sept.) 1933.

The six following preparations were used in this study: (1) bismuth sodium tartrate, (2) bismo-cymol, (3) bismuth salicylate, in oily suspension, (4) quinine bismuth iodide in oily suspension, (5) tartro-quiniodine and (6) bismuth tartrate. This included all the known types for intramuscular use, i. e., insoluble oily suspensions, a water-soluble compound, an oil-soluble compound and a preparation containing both a soluble and an insoluble salt, and a water-soluble salt for intravenous use. The doses and course of treatment are described.

The authors studied the effect of these preparations on (1) the resolution of cutaneous lesions, (2) the hepatic and renal functions, (3) the blood pressure, (4) the hemoglobin and red blood cells, (5) the Wassermann reaction of the blood, (6) the disappearance of spirochetes from open lesions and (7) complications occurring during treatment. This is a most interesting and instructive article. The authors conclude with fifty specific statements regarding the compounds used. The most significant is as follows: An oily suspension of bismuth is preferable to either a water-soluble or an oil-soluble preparation. A 10 per cent suspension of bismuth salicylate in a vegetable oil is the most effective preparation that we have had the opportunity to study, not only from the standpoint of therapeutic efficacy but also as regards the paucity of complications following its use. Despite the none too rapid absorption and the comparatively slow excretion of this compound, harmful accumulative tendencies were not observed. It is an ideal preparation for routine clinic use.

DULIN, Iowa City.

DIAGNOSTIC VALUE OF THE ORGANIC LUETIN REACTION. J. V. AMBLER, Arch. Dermat. & Syph. **28**:353 (Sept.) 1933.

The reports of several investigators are reviewed, and the author gives his results in a series of five hundred cases in which he used the intradermal test, injecting organic luetin prepared from the testes of syphilitic rabbits. He used a similar extract from normal rabbits for control. He obtained a false positive reaction in 1.9 per cent; only a small proportion of syphilitic patients showed a positive reaction to luetin. It was of greatest value in cases of syphilis of the skin and in congenital and ocular syphilis. The author believes that the present status of the test does not warrant its general use, but thinks that it may prove of value in ocular syphilis, as the reaction was 100 per cent positive in these cases and was negative in cases of interstitial keratitis and iridocyclitis which at first resembled syphilis but which proved to be tuberculous.

DULIN, Iowa City.

THERAPEUTIC EFFICIENCY OF BISMARSEN IN EXPERIMENTAL SYPHILIS IN RABBITS. GEORGE W. RAIZISS and MARIE SEVERAC, Arch. Dermat. & Syph. **28**:389 (Sept.) 1933.

This article compares the efficiency of bismarsen with that of arsphenamine. Bismarsen was synthesized by one of the authors (G. W. Raiziss) in 1924. He describes it as the result of an attempt to combine in one molecule two of the best spirocheticidal elements, arsenic and bismuth. It is the sodium salt of a bismuth derivative of arsphenamine methylene sulphonic acid. He reviews the studies of its clinical use and gives the results. They were favorable in all types of syphilis; in tabes dorsalis, in which it had a tonic effect and lessened the pain; in cardiovascular and acute syphilis in elderly patients; in the serologically negative stage of chancre; in congenital syphilis; in pulmonary spirochetosis; in severe Vincent's angina, and in acute meningeal syphilis. One group of workers reported a somewhat higher incidence of relapses with bismarsen than with other methods of treatment, and they considered that this offsets to some extent the favorable results obtained with the drug in a seronegative stage of chancre.

The remainder of the article gives in detail the authors' experimental studies on albino rats and rabbits. It was found that the albino rats tolerated a maximum dose of bismarsen of from 450 to 500 mg. per kilogram and that the rabbits could tolerate from 100 to 150 mg. per kilogram without toxic effects. The therapeutic dose was found to be from 10 to 15 mg. per kilogram for syphilitic rabbits, in contrast to from 16 to 18 mg. per kilogram of arsphenamine given intravenously and 20 mg. per kilogram when given intramuscularly. The effect of bismarsen in divided doses given weekly or semiweekly was also tested.

DULIN, Iowa City.

THE SPECIFICITY OF THE KAHN TEST IN MALARIA. C. R. SMITH, J. Lab. & Clin. Med. **18**:396 (Jan.) 1933.

It has been reported that a Wassermann test is not reliable in cases of acute malaria. Smith studied one hundred and ninety-four patients who were acutely ill with malaria and found that fifty-six of these showed a positive Kahn reaction. These fifty-six still showed a positive reaction after the malaria had cleared up. He concluded that the Kahn test is reliable, even though patients have acute malaria.

MCMURRAY, Galveston, Texas.

A ROUTINE CLINICAL EXAMINATION FOR TUBERCLE BACILLI IN MICROSCOPIC NEGATIVE SPUTUMS BY VARIOUS CULTURE METHODS. H. J. CORPER and W. L. COHN, J. Lab. & Clin. Med. **18**:515 (Feb.) 1933.

Of a series of one hundred sputums which were repeatedly negative for tubercle bacilli for a period of from four months to two years, 71 per cent were found

to be positive by culture on potato and egg yolk medium. It is suggested that culture methods be used more frequently in the diagnosis of tuberculosis.

McMURRAY, Galveston, Texas.

A CLINICAL AND STATISTICAL EVALUATION OF A NEW FLOCCULATION TEST FOR SYPHILIS BASED ON 26,611 TESTS IN COMPARISON WITH THE WASSERMANN REACTION, AND 2,473 IN COMPARISON WITH THE KAHN. H. EAGLE, J. Lab. & Clin. Med. **18**:684 (April) 1933.

In the author's experience, this test has proved more sensitive than either the Wassermann or Kahn tests. He detected 75 per cent of 10,383 known syphilitic serums, as compared with 58.7 per cent and 60.2 per cent detected by the Wassermann and Kahn tests. Also, this test shows less doubtful reactions—0.7 per cent, as compared with 1.4 per cent for the Wassermann and Kahn tests. The incidence of false positive reactions was lower than with the Wassermann or Kahn tests. A complete account of the technic and interpretation of the test is given.

McMURRAY, Galveston, Texas.

BISMUTH IN THE TREATMENT OF SYPHILIS. H. BECKMAN, J. Lab. & Clin. Med. **18**:1136 (July) 1933.

The author concludes that in congenital syphilis the use of bismuth alone is better than the use of mercury alone, but that bismuth is not as good as the arsphenamines. In some instances in which the Wassermann reaction remained positive after treatment with arsenicals and mercury a reversal was obtained with bismuth.

McMURRAY, Galveston, Texas.

AN EVALUATION OF THREE METHODS FOR THE DEMONSTRATION OF TUBERCLE BACILLI FOR USE IN HOSPITAL ROUTINE. N. HIRSCHBERG, J. Lab. & Clin. Med. **19**:429 (Jan.) 1934.

The author studied sixty-nine specimens by direct smear, inoculation of guinea-pigs and culture methods. By culture 24.6 per cent were positive, by direct smear 10.1 per cent and by inoculation of guinea-pigs 15.9 per cent. In one instance only inoculation of guinea-pigs gave positive results. In all other positive cases culture also gave positive results. The culture mediums recommended as a routine for hospital use are Corper's, Hohn's and Miraglia's. The latter two give more positive results than Corper's, but Corper's medium is less likely to become contaminated and for this reason should be used as a routine along with either Hohn's or Miraglia's medium.

McMURRAY, Galveston, Texas.

SYPHILIS AND PREMATURITY. ALWIN C. RAMBAR, J. Pediat. **3**:841 (Dec.) 1933.

This report consists of a study of one hundred and eighteen premature infants one or both parents of whom had a positive Wassermann or Kahn reaction. There are numerous references to the literature concerning syphilis and prematurity. Recently, acetarsone (stovarsol) has been the only drug used in treating syphilitic premature infants. This drug is preferred because of ease of administration and the resulting cooperation of the parents. Previous to the past year, mercury inunctions, mercury and chalk given by mouth and sulpharsphenamine given intramuscularly were employed. Rambar advocates at least two years of treatment for all premature offspring of syphilitic parents. It is considered inadvisable to wait for the onset of symptoms, because of the loss of valuable time in treatment and because so few syphilitic infants show definite signs for a period of months or even years. In this series early treatment resulted in a mortality of only 3.44 per cent of the infants discharged from the premature stations to their homes.

LEMASTER, Evanston, Ill.

MONOCYTOSIS AND VACCINATION WITH B C G. JULIO A. BAUZA, Bull. Soc. de pédiat. de Paris **30**:720 (Dec. 20) 1932.

Differential white blood counts were done on new-born infants. A group of normal infants showed a marked decrease in the number of neutrophils, a corresponding increase in the number of lymphocytes and a slight variation in the number of monocytes, in the first month of life. There was a slight increase in the number of monocytes in the first two weeks and a slight decrease in the third and fourth weeks. A second group of infants, who received B C G orally, showed a slight increase in monocytes over the control group in the third and fourth weeks. Fifty per cent of a third group of infants, who were given B C G subcutaneously, showed an increase in the number of monocytes, and after the second week the average number of monocytes in this group was greater than the average figure obtained in the control group. Because of the marked increase in lymphocytes in the normal infant during the first month, the lymphocytic variations in the two vaccinated groups are of no special significance.

BENJAMIN, Montreal, Canada.

INCREASE IN MORTALITY FROM TUBERCULOSIS AMONG CHILDREN IN 1931. L. BLUM, Gesundhfürs. f. d. Kindesalt. **8**:260 (Nov.) 1933.

For the first time since the beginning of the depression in 1931 an increase in mortality from tuberculosis has been noted among children. This increase is greater in the country than in the cities and is greatest in the regions where the percentage of jobless persons is highest. Other factors could be excluded, and the author believes that the influence of the economic depression has finally produced this result after two years.

REICHLE, Cleveland.

## Internal Diseases

CONGENITAL HYPERTROPHIC PYLORIC STENOSIS. E. H. SPIEGELBERG, Wisconsin M. J. **32**:297 (May) 1933.

The thickened wall of the stomach in congenital hypertrophic pyloric stenosis gives the vomitus its projectile character. There are dilatation and secondary gastritis which account for the traces of blood frequently seen in the vomitus. The etiology is still obscure. Because of the predominance in males it may be closely allied to hemophilia. With care, the pyloric tumor can be palpated in every instance.

Weaning and medical treatment are condemned. The mortality among the patients who are breast fed and operated on early is about 1 per cent. In the artificially fed infants it is three times greater.

Preoperative rehydration is urged. Ether is the anesthetic of choice. The stomach should always be emptied before the Ramstedt operation is performed.

JANNEY, Milwaukee.

THE NON-PROTEIN NITROGEN OF THE BLOOD IN HEALTH AND IN HEPATIC DISEASE. MARGARET T. TINDAL, Arch. Dis. Childhood **8**:17 (Feb.) 1933.

The nonprotein nitrogen of the blood after the ingestion of 15 Gm. of urea was investigated in twelve children free from hepatic disease and in fifteen children with hepatic disease. The curve for nonprotein nitrogen was found to be more prolonged in children with hepatic disease than in normal subjects.

KELLY, Milwaukee.

ATELECTATIC BRONCHIECTASIS IN CHILDHOOD. RICHARD W. B. ELLIS, Arch. Dis. Childhood **8**:25 (Feb.) 1933.

The triangular shadow situated behind the heart shadow on the left side or across the cardiophrenic angle on the right, as seen in x-ray films, was investigated.

Eight cases were studied by means of the injection of iodized poppy seed oil 40 per cent. Case histories and roentgenograms are given, and the author expresses the opinion that collapse of one or both lower lobes and failure to expand following pneumonia in infancy result in retention of bronchial secretions and bronchial dilatation. The longer the condition exists, the less is the chance for expansion of the lobe.

Clinically, there may be few symptoms of collapse of a lobe unless secondary infection takes place, and cough and fetid sputum may occur. Patients with pneumonia with persistent cough or signs in the chest should not be discharged until roentgen examination of the chest has ruled out collapse of a lobe.

Breathing exercises and inhalations of carbon dioxide are valuable in treatment. The author doubts that lobectomy is as yet advisable in cases of this type in childhood.

KELLY, Milwaukee.

KETOGENIC DIET IN THE TREATMENT OF PYURIA IN CHILDHOOD. J. BASIL RENNIE, Arch. Dis. Childhood **8**:47 (Feb.) 1933.

Six patients with chronic pyogenic infection of the urinary tract were treated with a ketogenic diet. At the end of treatment the urine was sterile in four cases, and growth on culture was scanty in a fifth; but in three of these cases there was a relapse within three weeks. The sixth patient showed no change in the condition. In the two patients who were permanently cured, the duration of the disease was short, and no abnormality of the urinary tract was observed. In each of the four cases in which treatment failed, abnormality of the urinary tract was demonstrated by pyelography. It would appear that a ketogenic diet is of little value as a curative agent in pyuria associated with abnormality of the urinary tract.

KELLY, Milwaukee.

THE HAEMOLYTIC STREPTOCOCCUS AS A FACTOR IN THE CAUSATION OF ACUTE RHEUMATISM. H. J. GIBSON, W. A. R. THOMSON and D. STEWART, Arch. Dis. Childhood **8**:57 (Feb.) 1933.

A study of 140 patients with acute rheumatism and 145 controls was made. Cultures were taken from the throat, and the intradermal reactions to extracts of streptococci of different types were ascertained. Strong positive reactions to hemolytic streptococci were more common in the rheumatic patients. Hemolytic streptococci were isolated from the throats of 43 per cent of the rheumatic patients and 20 per cent of the controls. The Dick test was positive in 16 per cent of the rheumatic patients and in 28 per cent of the controls.

The conclusion is reached that the allergic skin reaction may be a result of previous infection with hemolytic streptococci. The presence of cutaneous hypersensitiveness to intracellular antigens of this micro-organism cannot be regarded as an indication of the special reactivity necessary to produce acute rheumatism on infection. The intradermal reaction does not appear to be of direct diagnostic or prognostic value in cases of rheumatic infection.

KELLY, Milwaukee.

A CASE OF HEPATOMEGALY DUE TO VON GIERKE'S DISEASE. EDWIN C. WARNER, Lancet **1**:1070 (May 20) 1933.

The case reported is that of a girl, aged 2 years and 10 months, whose liver reached the level of the umbilicus. The van den Berg reaction was negative, and the levulose tolerance normal. The dextrose tolerance showed, one hundred and ten minutes after 112 mg. of dextrose had been administered, a value during rest of 86 mg. During the test, no sugar appeared in the urine, but a moderate quantity of acetone was present at the beginning of the test, and it was in greater quantity at the end of the test. The blood sugar during fasting was 48 mg., and there was no appreciable rise after a subcutaneous injection of 4 minims (0.24 cc.) of epinephrine, as compared with that of a control child of the same age.

It has been stated that the condition can be diagnosed only by hepatic puncture, but, as Schall pointed out, hypoglycemia readily occurs, accompanied by acetonuria, and epinephrine cannot mobilize the dextrose from the liver.

LANGMANN, New York.

SPECIMEN AND PHOTOGRAPHS OF CASE OF NEUROBLASTOMA OF THE SUPRARENAL GLAND (HUTCHINSON TYPE). J. V. C. BRAITHWAITE, Proc. Roy. Soc. Med. **26**:279 (Jan.) 1933.

The history, the results of physical examination, including laboratory examinations, the postmortem report and photographs of the child, the specimen and sections of the tumor are presented in the report of a case.

WILLIAMSON, New Orleans.

DIABETES INSIPIDUS AND ANTERIOR PITUITARY LOBE DEFICIENCY. R. W. B. ELLIS, Proc. Roy. Soc. Med. **26**:282 (Jan.) 1933.

A case was reported in a boy of 7 years. Until the age of 3 years he had been well except for an attack of measles. About that time he became drowsy and listless. He lost weight and began to suffer from increasing thirst and polyuria. The fundi and sella turcica appeared normal. The Wassermann reaction was negative; urinalysis showed a low specific gravity, with an average daily output of 4,095 cc. The patient was given pitressin (5 units twice a day) by hypodermic injection, which controlled the condition in spite of a steady gain in weight. After each injection there was a slight vasomotor disturbance. There was no history of headache or of visual disturbance. The author gives the results of physical examination and includes a photograph of the patient.

WILLIAMSON, New Orleans.

POSTERIOR MEDIASTINITIS WITH EFFUSION. R. W. B. ELLIS, Proc. Roy. Soc. Med. **26**:284 (Jan.) 1933.

The author reports a case of posterior mediastinitis with effusion in a girl of 7 years. He includes the history, the results of physical examination, temperature charts and the roentgenologic reports, with pictures of the x-ray plates taken from time to time during the patient's stay of eight weeks in the hospital.

WILLIAMSON, New Orleans.

GAUCHER'S DISEASE: SPLENECTOMY. BERNARD MYERS, Proc. Roy. Soc. Med. **26**:360 (Feb.) 1933.

The author reports a case of Gaucher's disease in a child, aged 3½ years, who before splenectomy had suffered from enlargement of the spleen and liver and had three attacks of epistaxis. There was marked anemia, the red cell count being as low as 1,500,000, with 19 per cent hemoglobin. After splenectomy the child had an attack of pneumonia and made a good recovery. She also suffered from osteomyelitis of the right femur. Sections made from necrosed bones showed Gaucher's cells. From this condition also she made a satisfactory recovery. Her blood, when last examined in January 1932, showed 4,500,000 red cells and 88 per cent hemoglobin. A photomicrograph of a section of the spleen is shown in the report.

WILLIAMSON, New Orleans.

RENAL DWARFISM: A REPORT OF TWENTY CASES WITH SPECIAL REFERENCE TO ITS ASSOCIATION WITH CERTAIN DILATATIONS OF THE URINARY TRACT. ARTHUR ELLIS and HORACE EVANS, Quart. J. Med. **2**:231 (April) 1933.

The material for this report consists of twenty cases of renal dwarfism studied by the authors in London. At the time of observation, the ages of the patients varied from 22 months to 26 years, twelve being under 15 years.

Observations on the urinary tract were available in seventeen instances, in fourteen of which there were varying degrees of dilatation. The urinary retention usually occurred at the level of the urethrovesical sphincter. No obvious cause for the obstruction was found. The authors suggested a disorder of the neuromuscular mechanism which controls the urethrovesical sphincter. The frequent association of renal dwarfism with this type of lesion is explained by the occurrence of long-persisting renal insufficiency. Arterial hypertension was not noted in these patients. Abstracts of the clinical records and several photographs of the patients are appended.

BRADFORD, Rochester, N. Y.

ROENTGENOLOGIC ASPECTS OF BRONCHOPNEUMONIA IN INFANCY. G. MOURIQUAND, M. BERNHEIM and J. SAVOYE, Arch. de méd. d. enf. **36**:265 (May) 1933.

The well known "triangle of hepatization," first described by Weill and Mouriquand, to distinguish lobar pneumonia from other types, has been accepted by most clinicians as pathognomonic.

To refute certain claims that bronchopneumonia in infants gives approximately the same roentgenologic findings, the authors made a careful roentgenographic study of 120 cases, several of which are reported in detail. They came to the conclusion that roentgenologic evidence in bronchopneumonia is extremely variable and cannot be utilized either for diagnostic or for prognostic purposes, as may the roentgenograms in lobar pneumonia.

AMESSE, Denver.

A STUDY OF INTESTINAL INFESTATION AMONG THE CHILDREN OF PARIS. P. NOBÉCOURT, C. BIDOT and A. COMNINOS, Arch. de méd. d. enf. **36**:329 (June) 1933.

Authorities differ as to the rôle of intestinal parasites in the production of disease; while such phenomena as infection, trauma, inflammation, irritation and toxic absorption occasionally may be charged to their activity, it is obvious that wholesale infestation may be present without the development of a single sign or symptom.

With the object of determining the relative frequency of parasitism in children in Paris, the authors conducted a careful study of the stools of 300 children at the medical clinic, among both the hospitalized children and the outpatients. It was found that infestation was extremely light during the first year, not exceeding 4.25 per cent, but this incidence increased rapidly with age, reaching 58.5 per cent at 5 years and 63 per cent at 16 years. There was apparently no difference from the standpoint of sex. The parasites encountered most frequently, particularly among children disposed to digestive disturbance, were tricocephali, ascarides, giardiae and spirochetes.

AMESSE, Denver.

THE INCIDENCE OF INFESTATION WITH OXYURIA VERMICULARIS AMONG RUSSIAN CHILDREN. Z. S. LEVINE, Arch. de méd. d. enf. **36**:344 (June) 1933.

A study of 404 children ranging in age from 7 to 15 years, conducted at Minsk, U. S. S. R., revealed an incidence of infestation of 51.2 per cent among males and of 48.8 per cent among females.

The diagnosis was made, not through the usual method of examination of the stools, but by smears from the folds of the perineum about the anus and from the external genitalia of females, together with careful investigation of material from the children's finger-nails. The elimination of infestation in schools consisted in hygienic and prophylactic measures, such as close trimming of the nails, washing the hands before meals and prevention of nail-biting and similar faulty habits of health. Medical treatment included topical applications of a solution containing

garlic every two or three days, after a cleansing bath. Santonin was also administered, and the author recommends the routine use of this remedy for all pupils in all types of school.

AMESSE, Denver.

MITRAL STENOSIS WITHOUT AUDIBLE MURMURS. H. GRENET and P. ISAAC-GEORGES, Bull. Soc. de pédiat. de Paris **30**:84 (Feb. 16) 1932.

A girl, 11½ years old, had repeated hemoptysis from the time that she was 8 years of age. She presented no abnormal physical findings. The diagnosis of mitral stenosis was made by roentgenologic examination.

BENJAMIN, Montreal, Canada.

THE RATE OF RED CELL SEDIMENTATION IN THE INFECTIOUS DISEASES OF CHILDREN. GRENET, ISAAC-GEORGES and PICHON, Bull. Soc. de pédiat. de Paris **30**:103 (Feb. 16) 1932.

In general, the rate of red blood cell sedimentation is increased in all the infectious diseases, in children as in adults. It recedes toward the normal at a slower pace than the clinical symptoms, especially the fever, and it increases slowly after exacerbations of the illness have been recognized by clinical observation. It is important to note that a coryza or ordinary cold or even a minor dental infection has been shown to increase the rate. Little is known of the influence of the many variable factors in the blood, such as the number of red cells, the surface tension of the plasma, the size of cells, the $p_H$, agglutinins, the carbon dioxide content and the bile salts. In the present state of knowledge, the sedimentation rate is merely a figure and not a measure of some known physiopathologic change. On the whole it is an objective determination which as yet does not yield any information which cannot be recognized, in most cases sooner, by clinical observation of the patient.

BENJAMIN, Montreal, Canada.

SEVERE BRONCHOPNEUMONIA IN AN INFANT OF NINE DAYS. ATTEMPT AT DIRECT VACCINATION THROUGH THE TRACHEA. CURE. M. LE LORIER, Bull. Soc. de pédiat. de Paris **30**:108 (Feb. 16) 1932.

Bronchopneumonia developed in a male infant on the sixth day after birth. Three days later he was so sick that the outlook appeared to be hopeless. Le Lorier introduced a tracheal aspiration tube into the trachea as far as the bifurcation and through it injected about 0.25 cc. of a stock vaccine "which contained some lytic substance." There was no immediate untoward reaction. The following morning the infant's condition was much improved, and forty-eight hours later he appeared to be normal. Le Lorier quite rightly remarks that it would be difficult to draw any conclusion regarding the therapy used from this one instance.

BENJAMIN, Montreal, Canada.

PSEUDOLEUKEMIC ANEMIA OF JAKSCH-HAYEM TYPE SUCCESSFULLY TREATED WITH LIVER EXTRACT. THE DIFFERENCE BETWEEN THIS FORM OF ANEMIA AND SIMPLE NUTRITIONAL ANEMIA OF THE CHLOROTIC TYPE. ROHMER, Bull. Soc. de pédiat. de Paris **30**:109 (Feb. 16) 1932.

A male infant presented marked pallor, malnutrition and marked enlargement of the liver and spleen at the age of 14 months. He had been declining since the age of 9 months. The blood picture was that of pronounced secondary anemia. He was given injections of liver extract and improved rapidly. Rohmer prefers to retain the term "pseudoleukemic" for this type of anemia, and to classify as "nutritional" only those cases of anemia which are cured by the administration of iron and vitamin C; cases in which the latter is needed, he believes, should be classed as cases of hypovitaminosis C until knowledge of the anemias of infancy becomes more exact.

BENJAMIN, Montreal, Canada.

BLOOD DISTURBANCE; EPISTAXIS REFRACTORY TO EVERY TREATMENT IN A GIRL ELEVEN AND A HALF YEARS OLD; SPLENECTOMY; IMMEDIATE CESSATION OF HEMORRHAGES. P. NOBÉCOURT, R. LIÉGE and GRODNITSKY, Bull. Soc. de pédiat. de Paris **30**:186 (March 15) 1932.

A girl, 11½ years old, showed numerous petechiae and had repeated severe nosebleeds. She was given a large number of treatments by mouth and subcutaneously, as well as blood transfusions, all without any lasting improvement. She became markedly anemic. After three preliminary transfusions, splenectomy was performed. She made an uneventful recovery. Within a few days after the operation, the bleeding time, which had been prolonged before, was normal, the blood platelet count, which had been low, was normal, and the clot retraction time, which had been delayed, also became normal. When the patient was observed three months later, these determinations were again abnormal, but there had been no more nosebleeds. Histologically, the spleen showed very little that was pathologic: the capsule was thickened, and there were slight congestion of the pulp and hyperplasia of the malpighian corpuscles. It is interesting to note that the child contracted malaria as a result of one of the transfusions. She was treated and recovered from the malaria. She also had diphtheria some time after the splenectomy, but showed no tendency to hemorrhage during the course of this disease, from which she made an uneventful recovery. The authors believe that splenectomy was a life-saving measure in this case, but whether it effected a permanent cure remains to be seen.

BENJAMIN, Montreal, Canada.

ATYPICAL SHADOWS OF THE HYPERTROPHIED THYMUS. P. DUHEM, Bull. Soc. de pédiat. de Paris **30**:194 (March 15) 1932.

Two anteroposterior roentgenograms of the chest of an infant, taken one month apart, are presented. A markedly increased hilar shadow on the right was at first thought to be due to enlarged tracheobronchial glands. Necropsy proved that it was caused by a hypertrophied thymus. Duhem states that the substernal space in an oblique view is usually clear in cases in which the widened anteroposterior shadow is due to adenopathy, whereas in those in which the thymus is hypertrophied, it is obscured.

BENJAMIN, Montreal, Canada.

SPONTANEOUS PYOPNEUMOTHORAX WITH SUBCUTANEOUS EMPHYSEMA IN A CHILD AGED TWENTY MONTHS, FOLLOWED BY CURE. M. CASSOUTE, POINSO and ZUCCOLI, Bull. Soc. de pédiat. de Paris **30**:206 (March 15) 1932.

A boy, 20 months old, suddenly became cyanosed and dyspneic. He presented swelling of the lower part of the neck and the upper part of the thorax due to subcutaneous emphysema. A button-hole incision was made in this region, and the child soon improved. There was no fever at this time. A few days later, high fever developed, and the child was again markedly dyspneic and cyanosed. Whereas a roentgenogram taken at the first examination showed nothing abnormal in the chest, a second one, taken after the onset of the fever, showed pyopneumothorax on the right side, some mottling and increase of the right hilar shadow and a patch of consolidation under the left clavicle. The child had a stormy course in the hospital, and on two occasions a fatal issue seemed imminent. During the second dangerous period the mother removed him from the hospital. About two and a half months later he was brought to the hospital in good condition, showing only mild bronchitis. A roentgenogram revealed only some increase in the right hilar shadow. The tuberculin reaction was repeatedly negative. Rupture of a subpleural pneumonic area in the mediastinal region was probably the cause of the subcutaneous emphysema and the pyopneumothorax. The pus was gradually evacuated from the pleural cavity by repeated vomiting.

BENJAMIN, Montreal, Canada.

A Case of Celiac Disease. P. Vallery-Radot, Bull. Soc. de pédiat. de Paris **30**:537 (Nov. 15) 1932.

A photograph of the profile of a little girl with celiac disease is published, illustrating enormous distention of the abdomen and the marked emaciation. The diarrhea began at the age of 18 months and was characteristically refractory to treatment. Since that time the disease has been running the typical irregular course of alternate periods of abatement and exacerbation. The author believes that celiac disease is rare in France.

Benjamin, Montreal, Canada.

A Case of Anterior Tibial Epiphysitis; Schlatter's Disease. Robert Debré, Robert Broca and Soulié, Bull. Soc. de pédiat. de Paris **30**:547 (Nov. 15) 1932.

A boy, 14 years old, had swelling of both tibial tubercles, with tenderness and some local increase of temperature. There was no history of trauma. Roentgenologic examination confirmed the diagnosis of Schlatter's disease.

Benjamin, Montreal, Canada.

A Case of Complex Osteopathy. G. Blechmann, A. Trèves and J.-T. Mornard, Bull. Soc. de pédiat. de Paris **30**:549 (Nov. 15) 1932.

A boy, 4 years old, presented multiple deformities of the bones. Roentgenologic examination showed marked decalcification of practically all the bones. The medulla of the femora particularly had a flocculent appearance with no visible trabeculation, and the cortex was extremely thin. The epiphyses were for the most part poorly developed. There was evidence of old fractures. The pathogenesis seemed to be complex. Certain features were suggestive of rickets, and others resembled achondroplasia. No definite diagnosis was made. No mention is made as to whether or not the corneas were cloudy. Dentition was markedly delayed, and the erupted teeth were very defective. Attempts at correction of the deformities by manipulation under anesthesia and the application of plaster casts were made.

Benjamin, Montreal, Canada.

Aplastic Anemia in a Child Improved by Injections of Bone Marrow Extract. Jules Milhit, Levesque, Papaioannou and J. Fouquet, Bull. Soc. de pédiat. de Paris **30**:553 (Nov. 15) 1932.

A girl, 5 years old, was extremely anemic and weak. Her condition had become much worse during the fifteen days prior to admission to the hospital. She was waxy in appearance. The liver was enlarged, the spleen was not palpable, and there was no enlargement of the lymphatic glands. The red cell count was 1,800,000 per cubic millimeter of blood, the white cell count was 6,080, and the hemoglobin measured 8 per cent; there were no nucleated red blood cells. Under liver therapy, first by mouth and then by injection, the child became progressively worse. Transfusions gave no improvement. She was then given bone marrow by mouth for a time, with some improvement. As she did not relish the bone marrow, she was given injections of an extract of bone marrow of a calf fetus containing 0.25 Gm. of fresh marrow per cubic centimeter. In the fifteen days during which bone marrow was given by mouth the red blood cell count increased from 800,000 to 1,500,000 per cubic millimeter and nucleated red cells appeared. In the following month, during which 1 cc. of the extract was injected daily, the red cell count increased to 4,000,000. The white cell count also increased to normal. During the course of an intercurrent respiratory infection there was marked leukocytosis. The possibility of the improvement being a temporary one is mentioned, but it is thought that bone marrow therapy has a distinct place in the treatment of this type of anemia.

Benjamin, Montreal, Canada.

THE IMPORTANCE OF CHANGES IN THE BLOOD CHEMISTRY IN THE COURSE OF HYPERPYREXIC SYNDROMES AFTER OPERATION OR SHOCK IN THE INFANT. G. PHÉLIZET, Bull. Soc. de pédiat. de Paris **30**:562 (Nov. 15) 1932.

A small series of cases of hyperpyrexia and pallor after infection and after operation and shock, with and without general anesthesia, were studied. The alkaline reserve was usually low and the blood urea and chlorides high; the urine generally showed a high concentration of urea and a slight increase in chlorides. Two normal infants were given an inadequate amount of water for four days in order to compare the chemical changes in their blood and urine with those of the postoperative cases. They were found to be practically identical, the only difference being that, whereas in the normal experimentally dehydrated infants the changes occurred over a period of days, in the postoperative cases they appeared very acutely, often in a few hours. Phélizet believes that the marked perspiration as well as increased loss of fluid by way of the lungs plays an important part in the dehydration of infants who suffer from postoperative shock. He thinks that it would be beneficial to administer increased quantities of water before and after operation to prevent dehydration, and, if the temperature rises sharply postoperatively, to inject parenterally dextrose solution and bicarbonate solution in an effort to reestablish the chloride equilibrium in the body.

BENJAMIN, Montreal, Canada.

UNUSUAL MYOPATHY. BABONNEIX and WILM, Bull. Soc. de pédiat. de Paris **30**:635 (Dec. 20) 1932.

A boy, 9 years old, showed generalized obesity and marked disturbance of gait as well as of posture. There was marked weakness of the muscles of the lower extremities and of the spine, with alteration of the reflexes and electric responses in the lower extremities. The intelligence was normal. The cranial nerves were not involved. The spinal fluid was under increased pressure, and showed globulin and 50 cells per cubic millimeter of fluid. The boy had begun to grow fat abnormally and to have difficulty in walking about two years previously.

BENJAMIN, Montreal, Canada.

SUFFOCATIVE GOITER. BABONNEIX and WILM, Bull. Soc. de pédiat. de Paris **30**:637 (Dec. 20) 1932.

A boy, 13 years of age, began to have dyspnea with the appearance of a marked swelling of the thyroid gland. His condition grew worse during a period of three days. He showed evidence of considerable pressure on the great vessels at the base of the heart and in the neck. Roentgenologic examination revealed a large shadow extending from the cardiac shadow into the neck, where it corresponded to the palpable enlargement of the thyroid gland. Exophthalmos, tremor and tachycardia were not present. He was given a treatment with roentgen rays; the same day the dyspnea subsided, and ten days later one could hardly palpate the thyroid gland. A week after this treatment the basal metabolic rate was plus 16; there is no mention of the rate before treatment.

BENJAMIN, Montreal, Canada.

ICTERUS WITH RELAPSE ASSOCIATED WITH INFECTION BY THE DIPLOBACILLUS OF FRIEDLAENDER. ROBERT DEBRÉ, F. CORDEY and DUCLOS, Bull. Soc. de pédiat. de Paris **30**:661 (Dec. 20) 1932.

A boy, 8 years old, was severely ill with jaundice and passed clay-colored stools and dark urine containing bile. A week after the onset of the illness, which was five days after the onset of the jaundice, a blood culture was taken, from which the bacillus of Friedländer grew. The boy's condition improved and the jaundice gradually disappeared, only to return, accompanied by severe illness, two weeks after its first appearance. A second blood culture was positive, yielding a

growth of the same organism as was previously identified in the blood. The boy gradually improved. Typhoid fever was excluded; the serum did not agglutinate Spirochaeta icterohaemorrhagiae, but did agglutinate Friedländer's pneumobacillus in a dilution of 1:360. It is considered that the latter organism may have been present as an accompanying infection and not as the etiologic factor in the pathogenesis of the acute hepatitis with jaundice. BENJAMIN, Montreal, Canada.

RHEUMATIC DISEASE WITH ESOPHAGEAL SYMPTOMS. P. ISAAC-GEORGES and P. DANSAERT, Bull. Soc. de pédiat. de Paris **30**:679 (Dec. 20) 1932.

A boy, 10 years old, had dysphagia for six months. The pain which he experienced on swallowing solid food was localized at the level of the spinous processes of the ninth and tenth dorsal vertebrae. A pericardial friction was heard. He was relieved of the pain when medication with a salicylate was tried. About a year previously he had complained of pain in the left knee which lasted for four days. It is thought that the dysphagic pain was periesophageal in character, caused by rheumatic mediastinopericarditis. BENJAMIN, Montreal, Canada.

VARIATIONS IN THE ARNETH NEUTROPHIL PICTURE IN INFANTS UNDER THE INFLUENCE OF THE HELIO-MARINE CURE. E. HURMUZACHE, Bull. Soc. de pédiat. de Iasi **3**:89 (Oct. 31) 1932.

Hurmuzache obtained the Arneth index on seventy children with glandular tuberculosis, aged from 12 to 15 years, before and after a month's rest at Bucacki on the Black Sea. Before treatment a decided shift to the left was found. Seventy-one per cent of the neutrophils had 1 or 2 nuclei, and 29 per cent had 3, 4 or 5 nuclei. After the month at the seashore the Arneth index showed a definite shift toward the normal. In 65 per cent of the neutrophils there were 1 or 2 nuclei and in 35 per cent 3, 4 or 5 nuclei. This shift toward the right coincided with a clinical improvement in the patients. LEMASTER, Evanston, Ill.

A CASE OF ESSENTIAL THROMBOPENIA. O. BARAS, Bull. Soc. de pédiat. de Iasi **3**:138 (Dec. 19) 1932.

A case of essential thrombopenia in a girl aged 13 years is reported. From the time that she was 5 years of age she had frequent epistaxis, excessive bleeding from minor wounds and large purpuric spots from slight trauma. The platelets numbered 60,000; the red blood cells, 2,100,000, and the white blood cells, 13,050. The bleeding time was two and one-half minutes but the coagulation time was five hours. Retraction of the blood clot did not occur until after five hours.

LEMASTER, Evanston, Ill.

HEPATOSPLENOGRAPHY. P. E. MORHARDT, Presse méd. **40**:1695 (Nov. 12) 1932.

Efforts to obtain roentgenologic delineation of the liver and spleen have been unsatisfactory heretofore, but thorotrast, containing 25 per cent of metallic thorium, gives a fairly good outline, with enough variation in density for pathologic conditions to be recognized. At times other organs, particularly the pancreas, may be outlined by the use of thorotrast. Contraindications to its use are diffuse pathologic changes in the spleen and liver, nephritis, jaundice, advanced cirrhosis, leukemia, hemorrhagic states and asthma. Unfavorable reactions, such as urticaria, edema glottidis, fever, vertigo, headache, chills and diarrhea, may occur in some cases. Thorium rapidly disappears from the blood stream, being deposited in the spleen and liver, whence its elimination is very slow. It exhibits a peculiar affinity for the reticulo-endothelial system. BIEKER, St. Petersburg, Fla.

SIGNS OF INFANTILE KALA-AZAR IN THE VASCULAR BLOOD. P. GIRAUD and R. POINSO, Presse méd. **41**:72 (Jan. 14) 1933.

Since kala-azar is a disease affecting the hematopoietic organs, the writers have studied a series of patients with the object of determining the effect on the blood and the capillaries. They have recorded the platelet count, the bleeding and clotting time, retractility of the clot and the tourniquet sign. Their findings are: The platelet count is generally diminished; the bleeding time, prolonged; the clotting time, normal; the retractility of the clot, normal and the tourniquet sign, positive. They warn against indiscriminate splenic puncture in the presence of such a hemorrhagic syndrome and advise that hemostatic medication be given as a preliminary measure if a puncture is to be made. Although antimony, is specific for the disease, it must be given with great caution in the hemorrhagic forms. Splenic irradiation is effective.

BIEKER, St. Petersburg, Fla.

ACUTE ENCEPHALITIS IN INFANTILE PNEUMONIA. G. MOURIQUAND, M. BERNHEIM and J. BOUCOMONT, Presse méd. **41**:211 (Feb. 8) 1933.

True encephalitis is rare as a complication following pneumonia. It must be distinguished from purulent processes, meningitis and evanescent cerebromeningeal symptoms of pneumonia.

Two cases are reported in infants who had no previous nervous disorders, and in whom, following pneumonia, definite evidence of intracranial accidents developed. One showed symptoms of a lesion of the pyramidal tract; the other, symptoms of thalamic and hypothalamic damage. A diagnosis of encephalitis seemed certain, although lumbar puncture was not done in either case. At no time were any meningeal symptoms in evidence.

BIEKER, St. Petersburg, Fla.

NEUROLOGIC SYNDROME IN GAUCHER'S DISEASE. R. MEYER, Rev. franç. de pédiat. **8**:559, 1932.

There are four neurologic symptoms in Gaucher's disease: (1) catatonicity, (2) vegetative dysfunction, (3) dyskinesia and (4) motor pyramidal dysfunction. The syndrome is so typical that a diagnosis can be made without puncture of the liver. The literature is well reviewed, and the relation to Tay-Sachs' and to Niemann-Pick's disease is discussed. Gaucher's disease seems to appear exclusively in the Aryan race. It is familial but not hereditary. There is no treatment of any value, but splenectomy, as well as injections of splenohepatic extracts and cerebrosides, should be considered.

SANFORD, Chicago.

APLASTIC ANEMIA IN INFANTS. L. MORQUIO and A. VOLPE, Rev. franç. de pédiat. **9**:153 (Feb.) 1933.

The authors present ten cases of myelotoxic aplastic anemia observed in their clinic (Montévidéo) during ten years (from 1922 to 1932). Eight cases are accompanied by full observations at autopsy. In six of the cases the duration was two or three months; in one case, one month, and in one, eight months. The characteristic process was profound anemia of unknown etiology. Clinically, no treatment was of any avail—the administration of liver or radium or roentgen radiation. In almost all of the cases there were purpuric phenomena, although they were very mild and might have passed unnoticed in some cases. One patient, however, showed such severe manifestations that the condition would unquestionably be classed as that described by Frank as *thrombopénie maligne*. Most of the patients had fever, and two, marked hyperthermia. Only one showed Vincent's angina. The blood always showed erythroleukothrombopenia. There was no evidence of hemolysis. The autopsy observations, which were complete, showed the condition in every case to be aplastic myeloid anemia. The authors believe that this form is somewhat similar to types of lymphatic leukemia, and that only careful hematologic study during life and complete examination of the bone marrow after death will differentiate the conditions.

SANFORD, Chicago.

CLINICAL CONTRIBUTION TO THE STUDY OF HEMOPHILIA IN INFANCY. FRANCISCO DE NICOLO, Clin. pediat. **15**:538 (July) 1933.

The author presents a detailed discussion of diagnostic measures, with emphasis on the clinical and hematologic factors in the various hemorrhagic syndromes and special reference to hemophilia. The several known theories of the pathogenesis of the disorder are given, with a detailed account of its symptomatology.

The author reports a case in a boy, aged 12½ years, with arthropathy in which it was impossible to establish the influence of any hereditary factor. Hematologic studies of the patient's mother revealed that the bleeding time was markedly prolonged, and on the basis of this claim, the existence of a hereditary diathesis is established.

A review of the more modern therapeutic measures used in this disorder is given, and the author concludes by stating that in his case opotherapy was of no value.

SIGNORELLI, New Orleans.

BLOOD TRANSFUSION IN INFANCY. A. GISMONDI, Prat. pediat. **11**:51 (Feb.) 1933.

This article is a review of the subject without the addition of new material. The author particularly discusses the indications for and the technic of transfusion in young infants.

DOOLEY, Kent, Conn.

A CASE OF "PURPURA ACUTISSIMA." M. ZAMBRUNI, Prat. pediat. **11**:116 (March) 1932.

The author presents a fatal case of acute purpura in a 4 year old child and offers a general classification of purpura.

DOOLEY, Kent, Conn.

UREA IN THE SALIVA OF THE CHILD. G. SCARPULLA, Riv. di clin. pediat. **31**:811 (July) 1933.

The concentration of urea in the saliva is parallel to that in the blood and is usually slightly less. There is no constant relation between the urea of the saliva and the urea of the urine.

In most diseases of children, the percentage of urea in the saliva is the same as that in the saliva of the normal child. In tuberculosis and a few other conditions a small rise is noted.

*Percentage of Urea in Various Conditions*

| | Blood, Per Cent | Saliva, Per Cent |
|---|---|---|
| Tuberculosis | 0.33 to 0.48 | 0.30 to 0.38 |
| Acute infections | 0.29 to 0.42 | 0.29 to 0.37 |
| Bronchiectasis | 0.44 to 0.56 | 0.30 to 0.50 |
| Tubular nephritis | 0.36 to 0.40 | 0.32 to 0.40 |
| Normal children | 0.20 to 0.36 | 0.24 to 0.38 |

In acute glomerular nephritis with a marked increase of the urea in the blood, the salivary urea is more or less increased, but it does not remain strictly parallel with the amount of urea in the blood, and in certain instances it has been definitely less.

HIGGINS, Boston.

THE EVOLUTION OF RHEUMATIC CARDITIS IN CHILDHOOD. PERLINA WINOCUR, Arch. argent. de pediat. **4**:157 (March) 1933.

Six cases are reported to illustrate the points that the author wishes to emphasize.

There is a febrile, inflammatory, hyposystolic phase which begins with insufficiency of the left ventricle due to rheumatic myocarditis. Intense dyspnea, pre-

cordial pain, cardiac enlargement, alteration of heart sounds and gallop rhythm are present. At times there is tachycardia with arrhythmia. Pericarditis may or may not be present. The liver is generally enlarged.

The antecedents are not necessarily rheumatic.

The development of the condition is rapid, with frequent intervals of improvement. Pulmonary conditions, such as pleuritis, serofibrinous pleurisy, pulmonary congestion and pulmonary edema, are often responsible for some of the fever.

Treatment with salicylates and cardiac tonics may or may not be of benefit. Generally the course is hopelessly bad.

Myocardial lesions are always present. The author expresses some doubt with reference to the unfailing value of salicylates in the treatment of rheumatic carditis.

SCHLUTZ, Chicago.

PULMONARY SCLEROSIS IN CHILDHOOD. JUAN CARLOS NAVARRO and ANTONIO F. AJA, Arch. argent. de pediat. **4**:250 (April) 1933.

The authors report this rather rare condition in three children, aged 7, 14 and 16 years, respectively. The process is chronic. Pulmonary consolidation, especially on the left side, is common. There is much thoracic deformity with retardation of the left hemithorax. The amplitude is diminished on the diseased side. The patient slumps toward the affected side.

There is considerable atrophy of the intercostal, pectoral and scapular muscles. All of these factors give a certain immobility to the side of the chest affected. A complete loss of thoracic elasticity results. There are bronchopulmonary symptoms with râles and, at times, pectoriloquy. All of these signs are persistent.

The heart is finally displaced to the left. Syphilis sometimes may be an etiologic factor. Chronic tuberculosis is sometimes present. The results of treatment are unsatisfactory.

SCHLUTZ, Chicago.

A CASE OF ADIPOSOGENITAL DYSTROPHY. J. M. DA ROCHA, Arch. de pediat. d. Rio de Janeiro **5**:53 (Dec.) 1932.

The patient was a boy 11 years of age. His weight was 47 Kg. (15 Kg. above normal); his height was 135 cm., and the circumference of the head was 60 cm., of the chest, 88 cm., and of the abdomen, 83 cm. Roentgenograms of the skull are shown. The characteristics are adiposity and small genitalia, with occasionally a low temperature and a low metabolic rate. The only treatment is operation, if the sellar tumor is accessible.

SANFORD, Chicago.

IRON IN THE TREATMENT OF ANEMIAS OF INFANCY. L. N. DA GAMA FILHO, Arch. de pediat. d. Rio de Janeiro **5**:190 (March) 1933.

The author details two cases of anemia, in children of 20 months and 10 years, respectively, caused by intestinal disorders following the administration of vermifuges. Of the different forms of therapy, he believes that he has obtained the best results with a 1 per cent solution of iron chloride. Graphs and tables showing the results of examinations of the blood are given.

SANFORD, Chicago.

CORTICAL PULMONARY HYDATID CYST: CYSTIC PLEURITIS. JOSE BONABA and JOSE A. SOTO, Arch. de pediat. d. Uruguay **4**:133 (April) 1933.

The possibility of cystic corticopleuritis must not be overlooked in rural children.

Absence of vomitus and purulent expectoration does not preclude the possibility of rupture of a pulmonary hydatid cyst. Vomiting may not actually be present The contents of the cyst may have been evacuated into the stomach.

In the case reported by the authors, pericystic bronchiectasis was present. The bronchi may play a rôle in corticopleuritis in the form of bronchiectatic processes.

Roentgen examination after the injection of iodized poppy seed oil 40 per cent will give the exact location of the cyst and also reveal the point of rupture into the stomach.

SCHLUTZ, Chicago.

COOPERATION IN THE STUDY OF DIABETES MELLITUS AND ITS ANATOMIC PATHOLOGY IN INFANCY. MARTINEZ VARGAS, Med. de los niños **34**:33 (Feb.) 1933.

Loss of weight, thirst and frequent urination should make the physician suspect diabetes mellitus. In infancy the predominant symptom is emaciation. In a child, aged 7 years, the crown of the molars, canines and incisors had oval and circular spaces where the enamel had disappeared because of acidosis. The treatment consisted in a suitable diet, alkalis, such as sodium bicarbonate, given both orally and hypodermically, and insulin.

Histologic examination of the tissues in one case studied revealed the following: In the pancreas there were an abundant, fibrinous exudate and other evidence of inflammation. In some areas no cells remained. The destruction of the glandular acini began from the outside and consisted of fatty necrosis and cellular necrosis. The same process invaded the islands of Langerhans. Both the internal and the external secretions of the gland are affected, the former to a greater extent. In the liver there were areas of fibrinoid necrosis, fatty infiltration and vacuolation of the protoplasm. The kidneys were slightly enlarged and congested, and the capillaries in the medullary zone showed an accumulation of leukocytes, which were all mononuclears. The suprarenal glands showed a slight increase in the trabeculae; the lymph glands were congested; the cardiac walls were thin, and the brain was slightly enlarged and congested, with edema of the pia mater. The most marked changes were found in the pancreas.

DWYER, Kansas City, Mo.

SCROFULOSIS. ENRIQUE SUNER ORDONEZ, Pediatría españ. **21**:883 (Nov.) 1932.

The author is of the opinion that the present conceptions regarding scrofulosis are not much different from those held by the older writers. The clinical picture of this constitutional dyscrasia is well established.

Protein therapy in the form of horse serum, the pseudoglobulins or casein preparations is highly recommended. The author believes that by this procedure immunity against tuberculous infection is definitely assured. He is opposed to treatment of this dyscrasia with ductless gland preparations.

SCHLUTZ, Chicago.

GENERAL ECHINOCOCCOSIS. E. SISTO HONTAN, Pediatría españ. **22**:1 (Jan.) 1933.

The case reported occurred in a child 11 years old. Multiple echinococcic cysts were found in the abdomen and in the thorax. The thoracic cysts were both cortical and central. The location of the original cysts was in the liver. The abdominal cysts were removed by laparotomy. There are definite limitations in the treatment of pulmonary cysts. The author believes that those having a cortical location should be treated surgically and extirpated, whereas those located centrally should be treated entirely by aspiration.

SCHLUTZ, Chicago.

THE PRESENCE OF STREPTOCOCCI IN APICAL GRANULOMAS. W. BLUMENBERG and A. ZÜLL, Arch. f. Hyg. **109**:297 (Feb.) 1933.

Fifty-four apical granulomas were studied. Hemolytic streptococci were found in 50 per cent of the cases. The striking difference between these results and those of other investigators probably depends on the methods used. Geographic location and climate are probably factors, but their evaluation is difficult. The usual opinion that hemolytic streptococci are present only in granulomas manifesting

clinical symptoms is wrong. Hemolytic streptococci, usually in mixed culture, may be present independent of the clinical form of the granuloma. These latent foci of streptococcic infection should be eliminated. All the evidence indicates that septic conditions of dental origin are far more frequent than is generally supposed.

GLASER, Rochester, N. Y.

ABDOMINAL PAIN IN CHILDHOOD. JOSEF SIEGL, Arch. f. Kinderh., 1933, supp. 3.

The differential diagnosis of conditions causing abdominal pain in infancy and childhood is discussed from the clinical point of view. The proper technic for examining the child is carefully outlined. Emphasis is placed on the peculiarities and variations in the manifestations of diseases in early life, as contrasted with their occurrence in adults. Observation of the appearance and behavior of the child is of great importance in evaluating the significance of the child's complaints.

Appendicitis must always be considered when abdominal pain is present in children. Among the other conditions discussed are pyelitis, pneumonia, intussusception, strangulated hernia, osteomyelitis, gastro-enteritis and functional disturbances. The physical findings in these conditions and the clinical approach in distinguishing between them are presented very thoroughly and completely.

LEMASTER, Evanston, Ill.

PATHOLOGIC ANATOMY OF BANG'S DISEASE IN MAN. F. WOHLWILL, Virchows Arch. f. path. Anat. **286**:141, 1932.

The author briefly reviews the results of autopsies on patients who are said to have suffered from undulant fever. Up to the time of writing his paper, twenty-six cases had been reported in the literature, and he adds four cases of his own in patients who had died at St. George's Hospital (Hamburg) during the last three years. In only two of his cases was a complete autopsy performed. The first patient, who suffered from Laënnec's cirrhosis and Bang's disease, showed nothing which differed from the usual findings in the former disease. The second patient was a 67 year old woman who suffered from adipositas and diabetes. She had a temperature of 39 C. (102.2 F.) four days prior to her admission to the hospital. She presented a "typhoid-like" picture. The blood showed 3,900 white cells, with slight lymphocytosis. The spleen and liver were palpable. The fever was of an undulant type up to the time of the patient's death from embolism of the lungs, which occurred on the twenty-second day after hospitalization. The agglutination titer for Bang's bacillus was 1 : 800 on admission and rose to 1 : 3,200 three days before death. At autopsy, embolism of the common trunk due to thrombosis of the femoral vein was found. Macroscopic examination of the organs did not reveal any specific changes. Histologically, however, nodules containing giant cells were found in the spleen, liver, bone marrow and lymph nodes. These nodules consisted chiefly of reticulo-endothelial cells, which were transformed into light epithelioid cells, and some eosinophilic granulocytes and plasma cells. The nodules showed no tendency to caseate but contained considerable quantities of fat. The author discusses similar findings by others and the histologic picture of the experimental and spontaneous disease in animals. He thinks that these nodules belong to the infectious granulomas and that they represent a reaction of the reticulo-endothelial system to a general infection.

SCHOENTHAL, New York.

## Nervous Diseases

CEREBRAL LOCALIZATION OF EPILEPTIC MANIFESTATIONS. WILDER PENFIELD and LYLE GAGE, Arch. Neurol. & Psychiat. **30**:709 (Oct.) 1933.

This is a study of seventy-five cases of focal epilepsy. In nearly all cases cerebral pneumography was carried out, with the patient under local anesthesia. Seizures studied were spontaneous or induced by hyperpnea, hydration or direct cortical stimulation. Parts of the authors' summary are: "The most frequent

lateralizing sign is deviation of the head and eyes to the side opposite the hemisphere involved. In seizures which arise in the precentral or postcentral gyrus, consciousness is usually lost late. Consciousness is likewise apt to be lost in seizures arising anywhere behind the central sulcus, and such seizures are ushered in by auras. It must be remembered, however, that a major attack may leave retrograde amnesia, so that the aura is forgotten. Seizures originating in the supramarginal gyrus are characterized by a discontinuous twinkling of lights seen in the contralateral field without any involvement of the calcarine zone.

"An aura of pain or of epigastric distress may arise from activity of the cerebral cortex, and cortical stimulation reproduces such sensory phenomena. The buzzing sounds and the dizziness which are characteristic of unilateral temporal lobe seizures have been reproduced by electrical stimulation, but the more complicated dream states and odors have never been reproduced, perhaps because of the limitation of surgical approach.

"Electrical exploration of the cortex in conscious patients during craniotomy has given unusual opportunity to study cerebral physiology, both normal and abnormal."

BEVERLY, Chicago.

MEGALENCEPHALY WITH DIFFUSE GLIOBLASTOMATOSIS OF THE BRAIN STEM AND THE CEREBELLUM. ARTHUR WEIL, Arch. Neurol. & Psychiat. **30**:795 (Oct.) 1933.

A case of megalencephaly in which the brain weighed 1,856 Gm. is described in a boy, 7 years old, whose development was normal until the sixth year of life. Hypertrophy of the cerebral hemispheres was combined with diffuse glioblastomatosis of the brain stem and cerebellum. A living brother has a large head, which suggests congenital familial maldevelopment.

BEVERLY, Chicago.

VERIFIED TUMOR OF THE TEMPORAL LOBE. A CRITICAL REVIEW OF FIFTY-TWO CASES. STUART N. ROWE, Arch. Neurol. & Psychiat. **30**:824 (Oct.) 1933.

Rowe reviews fifty-two cases of verified tumor of the temporal lobe. This portion of the brain seems destined to remain a more or less silent area. Usually the advent of increased intracranial pressure produces the first symptoms to attract attention. Mental abnormalities varying from changes in consciousness to mild alterations in personality are frequent. Epilepsy was an important feature in 36 per cent of the cases. Aphasia was found in only one third of the cases with tumor on the left side. Hallucinations of taste and smell were present in six cases. Visual fields were of great diagnostic importance in 57 per cent of the cases. Information from ventriculography is helpful.

BEVERLY, Chicago.

THE PREVENTION OF NEUROSES IN CHILDREN. BERTRAND S. FROHMAN, Arch. Pediat. **50**:502 (July) 1933.

I see no reason to abstract this article.

WADDELL JR., University, Va.

MENINGOCOCCIC MENINGITIS IN INFANCY. A. BROWN and N. SILVERTHORNE, J. A. M. A. **101**:272 (July 22) 1933.

Brown and Silverthorne report a case of meningococcic septicemia with meningitis in an infant aged 22 days. A diagnosis was correctly made within twenty-four hours after the onset; adequate serum therapy was given immediately by intravenous and intrathecal routes, and cure occurred in fifteen days.

BONAR, Salt Lake City.

PRENATAL DEVELOPMENT OF BEHAVIOR IN THE ALBINO RAT. A. W. ANGULO Y GONZALEZ, J. Comp. Neurol. **55**:395 (Aug.) 1932.

This study was carried out on 643 fetuses obtained from 77 litters. They ranged in age from 14 to 21 days (term). The first sign of motility was found at fifteen days and eighteen hours after insemination. The movement consisted of a slight bending of the head, involving only the region of the neck and taking place only after stimulation of the region of the snout. The snout came closer to the stimulator, and hence this was not classified as an "avoiding reaction." Spontaneous activity was observed in several fetuses 16 days old. In the older fetuses motility spreads in the cephalocaudal and proximodistal direction. Thus, the movement progresses so as to involve first the forelimbs, later the rump and finally the hindlimbs. During the first stages of development the appendages move only with the trunk. This reaction of the total mass is called a "total pattern reaction." The total pattern reaction consists of a primary or basic movement, lateral flexion of the trunk and a series of secondary movements. Similarly there develops later a total pattern consisting of a basic movement of extension of the head and a series of secondary movements. During the latter period of development of fetal behavior a number of specific reflexes show what at first seems to be a breaking up of the total patterns into individual and specific reflexes. However, Gonzalez believes that the process by means of which the individuation and specificity of certain reflexes are attained is not a disintegration or breaking up of the established pattern, but an inhibitory action by means of which the primary movements are in a large measure arrested. In other words, the total pattern reaction is never abolished completely; nor is the dominance of the primary on the secondary movements lost.

The discussion of questions arising from the results of this investigation, with references to the work of others on kindred subjects, has been deferred to a later time.

FRASER, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

HEREDITARY ATAXIA. ALLAN PENNY BLOXSOM, J. Pediat. **3**:623 (Oct.) 1933.

A case of hereditary ataxia is reported. The onset followed an acute infectious process which occurred when the child was 2½ years old. Bloxsom favors the view that in this condition the nervous system is congenitally defective, and that the acute infectious process serves only to initiate the degenerative processes. The general subject of Friedreich's ataxia is discussed briefly.

LEMASTER, Evanston, Ill.

CHOREA IN THE NEGRO RACE. HAROLD C. LUETH AND DON C. SUTTON, J. Pediat. **3**:775 (Nov.) 1933.

Of 165 children studied in the clinic for the follow-up of patients with cardiac disease of the Cook County Hospital, 131 were white and 34 were Negroes. Chorea was observed in 37 per cent of the white children and 26 per cent of the colored children. Chorea showed the same characteristics in both races. These figures are published to refute the common statement that chorea is rarely seen in the Negro race.

LEMASTER, Evanston, Ill.

NEUROLOGICAL CONDITIONS IN INFANTS AND CHILDREN. R. CANNON ELEY, J. Pediat. **3**:781 (Nov.) 1933.

In this critical review Eley discusses recent contributions on the following diseases: narcolepsy, microcephalus, mongolism, poliomyelitis and tuberculous meningitis.

LEMASTER, Evanston, Ill.

MENTAL DEFICIENCY AMONG EDINBURGH SCHOOL CHILDREN. W. GEORGE SYM, Tr. Edinburgh Med.-Chir. Soc., 1933, p. 117; in Edinburgh M. J., July 1933.

Sym defines the term "mentally defective," describes the methods of ascertaining the condition, gives the number of such children and outlines what their future care should be. He states that he uses the intelligence quotient constantly in his work in Edinburgh. There are four schools for the mentally defective in that city The details of running such schools are mentioned. The teachers must have special training in hand-work, games, sewing, dancing and other types of occupational therapy for the defective child. Swimming is popular with the boys and girls in these schools.

Regarding the maturity of the children, Sym states that the average mentally defective boy is not nearly so likely to become a parent as the girl, for he cannot earn a livelihood sufficient to enable him to marry, while the girl may be easily led astray by a man of more intelligence.

NEFF, Kansas City, Mo.

MONGOLISM WITH SAGITTAL FRONTAL PROMINENCE. BABONNEIX and RIOM, Bull. Soc. de pédiat. de Paris **30**:85 (Feb. 16) 1932.

A boy, 3 years old, presented the features of mongolism. In addition he had a cranium shaped like a keel. This prominence at the site of the median suture of the frontal bone has been described as one of the stigmas of congenital syphilis. In this case there was no evidence of syphilis.

BENJAMIN, Montreal, Canada.

AMYOTROPHIC LATERAL SCLEROSIS IN A GIRL OF SIXTEEN YEARS. MARCEL LELONG, JEAN LEREBOULLET and FELIX-PIERRE MERKLEN, Bull. Soc. de pédiat. de Paris **30**:88 (Feb. 16) 1932.

This condition is rare in children, only four authentic cases in persons under 15 years of age having been reported. The child had marked atrophy of the small muscles of both hands, which were held as in Duchenne-Aran disease, bilateral footdrop and some defect in speech. The third, seventh and eighth cranial nerves were involved. There was no sensory involvement. Examination of the blood and spinal fluid gave negative results.

BENJAMIN, Montreal, Canada.

DOUBLE OBSTETRICAL BRACHIAL PARALYSIS. L. BABONNEIX and RIOM, Bull. Soc. de pédiat. de Paris **30**:154 (March 15) 1932.

A boy, 7 years old, showed marked motor and sensory impairment of both upper extremities, in spite of persistent treatment since a few days after birth, when the condition was first noticed.

BENJAMIN, Montreal, Canada.

PARALYSIS OF THE LEFT ARM FOLLOWING CARBON MONOXIDE POISONING. JEAN HALLÉ and JACQUES ODINET, Bull. Soc. de pédiat. de Paris **30**:157 (March 15) 1932.

A boy, 3 years old, was one of a family all the members of which were subjected to the fumes which emanated from a poorly set up stove which was lighted for the first time one cold winter night. The child was discovered in a coma after he had been in this atmosphere for about twenty hours. His head was found to be caught between two bars of his crib, one of which was pressing on his left clavicle and the other on the right side of his neck. He was dyspneic and cyanosed, and when he was revived it was noticed that he had left brachial plexus paralysis. It is believed that the latter was caused by carbon monoxide poisoning, although the compression of the right clavicular region is considered as a possible etiologic factor.

BENJAMIN, Montreal, Canada.

TETANY OF THE NEW-BORN CURED BY INJECTIONS OF PARATHYROID EXTRACT. H. DUFOUR and BARNEVELD, Bull. Soc. de pédiat. de Paris **30**:171 (March 15) 1932.

A girl who was born at full term and who weighed 4,000 Gm. at birth began to have tonic convulsions when she was 8 days old. She was at first thought to be suffering from tetanus and some antitetanic serum was administered. The diagnosis made by those reporting the case was tetany of the new-born. Small doses of calcium chloride were given, with subcutaneous injections of parathyroid extract, one a day for the first three days and then every other day until eleven were given. The infant had a syncopal seizure soon after one of the earlier injections; it is not known whether this was due to the illness or to the treatment. The condition was entirely cured at the end of three weeks. A determination of the calcium content of the mother's milk showed it to be higher than the average.

BENJAMIN, Montreal, Canada.

THE TREATMENT OF POLIOMYELITIS: A SEVERE CASE TERMINATED BY COMPLETE FUNCTIONAL CURE. PAUL GIRAUD and HUGUET, Bull. Soc. de pédiat. de Paris **30**:579 (Nov. 15) 1932.

A boy, 4 years old, had "complete paralysis, and could move only his eyes." There is no mention made of respiratory difficulty. A year later he could move all his muscles practically normally; there remained only some impairment in response to electric stimulation of a few muscles. He was given a wide range of treatments, both during the acute stage of the disease and throughout the year following, and it would be difficult to attribute the good recovery to any particular form of therapy used. It is believed, however, that the large doses of convalescent and prepared serum which were given saved his life, even though the first injection of serum was administered after the onset of paralysis.

BENJAMIN, Montreal, Canada.

THE PROFILE OF DEVELOPMENT IN THE PRACTICE OF NEUROPSYCHIATRY OF CHILDREN. GEORGES D' HEUCQUEVILLE, Hyg. ment. **28**:101 (May) 1933.

The author presents a technic for the neuropsychiatric examination of children. The technic was elaborated to conform with the following criteria: (1) that it be readily accessible to all practitioners; (2) that its administration require only a limited amount of time, from ten to fifteen minutes; (3) that it require no formal manual and permit of immediate interpretation and simple notation of results; (4) that it consist solely of facts of clinical interest, especially with reference to diagnosis, prognosis and treatment.

The proposed technic combines two principles, for it employs the graphic representation of a developmental profile or curve of the various stages of development of certain structural and functional traits expressed in terms of age levels or age increments. Seven tests are given. Three of these (age, weight and height) permit a qualitative appraisal of the subject, whereas the remaining four (rapidity of hammering, tapping test, verbal precision and memory) give a quantitative evaluation of the functional development of the nervous system.

The results are plotted in the proper columns of a graphic form and the points joined by a broken line, thus constituting a profile of development which, based on age values, gives the essential characteristics of the physical and behavior growth of the child. One is then able to determine whether the subject is of average, superior or inferior development as compared with the average for his age.

ABRAMSON, New York.

THE BABINSKI PHENOMENON IN INFANTS. M. SCHACHTER, J. belge de neurol. et de psychiat. **33**:180 (Feb.) 1933.

Schachter limits himself to a discussion of the literature, quoting from numerous papers. He believes that until the age of 2 months there is no distinct reaction;

from 2 to 3 months, reactions vary from dorsal to plantar flexion of the toes; from 3 to 4 months, dorsal flexion and fanning of the toes occur with occasionally the dorsal flexion changing to plantar flexion at the end of the reaction; from 4 to 6 months, a similar movement of the toes is noted, but is accompanied by a reflex which draws the leg toward the abdomen. The consistent reaction of plantar flexion occurs, according to various authors, between 3 months and 7 years, and after this period the appearance of the primitive formula indicates a pathologic lesion.

In the author's series of patients tested at the age of 5 months there was extension, but the extensor response was never pure, being associated with an added flexion or with extension and fanning to which was often added flexion of the foot and leg. The response was more active while the child was asleep. Excitation of cutaneous areas away from the sole in several of the cases caused extension of the great toe. This was most marked in a premature child aged 7 months. The author believes that before the age of 2 years the Babinski sign is not an evidence of pathologic change.

WAGGONER, Ann Arbor, Mich. [ARCH. NEUROL. & PSYCHIAT.]

THE POSSIBILITY OF PROVOKING THE PASSAGE OF ANTIBODIES INTO THE CEREBRAL SPINAL FLUID, WITH A TENTATIVE THERAPEUTIC APPLICATION IN HEINE-MEDIN DISEASE. L. AURICCHIO and M. DEL GIUDICE, Pediatria **41**:713 (June 1) 1933.

In a series of observations on rabbits the authors established that, having produced a provocative agglutination for typhoid, paratyphoid and melitensis bacilli in the blood, a like agglutination did not occur when the method was applied to the cerebrospinal fluid. Following the spinal injection of normal horse serum or of a colloidal metal, however, agglutination took place in the spinal fluid as well as in the blood. Because of these observations and the consideration that in acute anterior poliomyelitis the blood possesses high virucidal powers which are absent in the cerebrospinal fluid, the authors are convinced of therapeutic possibilities in spinal administration of either normal serum or a colloidal metal. This conviction caused them to apply such treatment to two patients (one 16 months and one 13 months of age), using 2 cc. doses of normal serum in the first and of colloidal metal in the latter. In each the clinical response was surprisingly satisfactory. Further observations of this type will be practiced and later reported.

SIGNORELLI, New Orleans.

CEREBRAL ABSCESS WITH CHRONIC EVOLUTION AND SIMULATING A HYDATID CYST. A. RODRIGUEZ CASTRO and H. CANTONNET, Arch. de pediat. d. Uruguay **4**:183 (May) 1933.

This condition occurred in a child 12 years old. Cephalalgia and nausea were prominent symptoms. There were marked intracranial pressure and enormous papillary edema with optic atrophy. An operation did not reveal the abscess, which was found at autopsy. The pus showed staphylococci.

SCHLUTZ, Chicago.

LUMBOSACRAL MENINGOCELE. J. GARRIDO-LESTACHE, Pediatria españ. **22**:318 (May) 1933.

The author reports a case of massive lumbosacral meningocele completely healed by operation in a child 4 years old. The technic advocated by Ribera and Bayer was followed. In suitable cases the author advises the use of bone grafts according to the method advocated by von Schmieden.

SCHLUTZ, Chicago.

EFFECT OF ROENTGEN IRRADIATION ON THE CHOLESTEROL OF THE BLOOD IN MONGOLIAN IDIOCY. A. MADER and ERNST BINGENHEIMER, Jahrb. f. Kinderh. **138**:9 (Jan.) 1933.

The level of cholesterol in the blood is low in mongolian idiocy. Marked elevation in the level occurs after roentgen irradiation, especially of the head. Repeated exposures appear to cause greater elevation. The appearance and the growth of the patients were not improved, but they seemed to become more alert and less irritable and appeared to resist infections better. No report is made of the effect of roentgen irradiation on the blood cholesterol in other conditions.

CLAUSEN, Rochester, N. Y.

RÔLE OF THE HEMATO-ENCEPHALIC BARRIER IN THE GENESIS OF INTOXICATION: VIII. HISTOLOGIC CHANGES IN THE CENTRAL NERVOUS SYSTEM. S. J. SCHAFERSTEIN, NINA POPOWA and EUGENIE OWTSCHARENKO, Jahrb. f. Kinderh. **138**:95 (Jan.) 1933.

In six cases of alimentary intoxication the authors found the pia mater edematous, hyperemic and infiltrated by lymphocytes. The choroid plexus was hyperemic and hemorrhagic and the epithelium degenerated. Degeneration of cells was observed in the cerebellum and the cerebral cortex, and especially in the tuber cinereum. The latter changes may give rise to disturbance in water metabolism.

CLAUSEN, Rochester, N. Y.

SYMPTOMS AND PATHOGENESIS OF CHOREA MINOR. OTTO KRÖNIG, Jahrb. f. Kinderh. **138**:160 (Feb.) 1933.

A study of forty cases of chorea minor led the author to the view that this disease results from invasion of various parts of the central nervous system by a virus.

CLAUSEN, Rochester, N. Y.

PATHOGENESIS OF INFANTILE TETANY. E. ROMINGER, HUGO MEYER and C. BOMSKOV, Klin. Wchnschr. **10**:1342 (July 18) 1931.

These authors consider infantile tetany in rachitic children an accompaniment of the beginning of the healing process (*Heilkrisis*). It consists in a one-sided, phosphorus remineralization without sufficient concomitant calcium mineralization. The rachitic organism at the beginning of healing robs the tissues of calcium for deposition in the bones, impoverishing the tissues and the blood (hypocalcemia). As healing proceeds, there comes again a deposition of phosphorus followed by a similar deposition of calcium.

The central point in the pathogenesis is the calling forth of phosphorus through the sudden beginning of healing. This leads to the lessening of ionized calcium. It also produces alkalosis. There is a sudden laying down of new bone before there is sufficient retention of calcium. There are thus hypocalcemia, hyperphosphatemia and alkalosis, and it can be understood why tetany is not dependent on the degree of hypocalcemia alone.

Besides the accumulation of phosphorus, other factors may produce a tendency to tetany. Alkalosis may arise not only from the rise in phosphorus but also from fever or from hyperventilation. There may also be factors influencing the calcium metabolism such as toxic substances or insufficient calcium intake or hormonal disturbances. These factors may produce tetany without the presence of the hyperphosphatemia of rickets.

SILVERMAN, Syracuse, N. Y.

ASEPTIC TRAUMATIC MENINGITIS. O. BOUET, Hospitalstid. **76**:793 (July 20) 1933.

In Bouet's three cases, symptoms of meningitis developed in from twenty-four to forty-eight hours after trauma to the head. The spinal fluid showed polymorpho-

nuclear leukocytes in two cases, and monocytes in one, but no bacteria. The fluid was under increased pressure. Repeated lumbar drainage relieved the symptoms, and recovery followed. Several cases of benign meningitis following injury to the head are cited from the literature.

ELGHAMMER, Chicago.

ENCEPHALITIS IN A CHILD SIMULATING COLIBACILLOSIS. S. M. C. VAN VEEN, Maandschr. v. kindergeneesk. **2**:419 (June) 1933.

The author describes a case of probable encephalitis which took the clinical course of a toxicosis. The question is raised whether the toxicosis was a complication of the encephalitis. Analogous cases from the literature are mentioned.

VAN CREVELD, Amsterdam, the Netherlands.

## Diseases of the Eye

TWO CASES OF BUPHTHALMUS IN SIBLINGS. C. HYMES, Am. J. Ophth. **17**:132 (Feb.) 1934.

Two cases of buphthalmus in sisters are reported. In the first the eyes were apparently normal up to the age of 6 months, at which time the parents observed that they were becoming enlarged. The right eye was operated on unsuccessfully and was phthisical. The left eye showed a steamy cornea, 17 mm. in diameter; the anterior chamber was deep and to palpation the tension appeared elevated. An Elliot trephining was done on this eye, following which the fundus could be seen, the optic nerve showing a deep glaucomatous cup. In the second case the parents noted that at the age of 7 months each cornea became hazy and the eyes were enlarging, as had happened in the older sister. Examination revealed enlarged eyeballs and corneas, the latter being steamy. Tension to palpation was increased. Trephining was done on both eyes, following which the corneas cleared. About six years after operation the first patient had vision of 5/200 in the left eye, and the second, vision of 20/40 in the right and 20/20 in the left eye. Hymes believes that patients showing buphthalmus should be operated on early.

REESE, Philadelphia. [ARCH. OPHTH.]

TRACHOMA IN THE WHITE POPULATION OF THE UNITED STATES. C. E. RICE, J. Missouri M. A. **31**:73 (Feb.) 1934.

The cases of trachoma studied were those of persons whose ancestors have been in this country for one hundred years, not those of emigrants or Indians. These persons are, for the most part, unable to employ a private physician or specialist and so are cared for by the state in cooperation with the United States Public Health Service.

In Rolla they are hospitalized, forty at a time, in a converted residence. The average stay in the hospital is twenty-nine days. The program consists of an educational campaign as to hygienic habits, and individual cases are located and treated. Field nurses are invaluable in all phases of this work.

Within the past ten years, more than 5,000 cases of trachoma have been contacted in the state of Missouri; most of the patients live south of the Missouri River. A high percentage of them are blind or nearly blind. Seven hundred persons in Missouri are receiving pensions because of blindness due to trachoma.

Similar hospitals are maintained in Kentucky and Tennessee.

BERGER, Kansas City, Mo.

THE MENACE OF HEREDITARY BLINDNESS. J. MYLES BICKERTON, Brit. M. J. **1**:93 (Jan. 20) 1934.

The author discusses blindness from all causes. He gives statistical data concerning the incidence of blindness, although he admits that these are imperfect, since registration of the blind is difficult. He places syphilitic conditions in early

life in the class of "pseudoheredity" conditions, as due to prenatal infection. Then follows a brief outline of all of the genetic types of blindness, with charts showing the familial incidence. He concludes with suggestions as to the possible remedy, among which sterilization of the unfit is given a prominent place.

ROYSTER, University, Va.

PARALYSIS OR CONGENITAL ABSENCE OF ACCOMMODATION IN THREE BROTHERS. J. SEDAN and A. ROUX, Ann. d'ocul. **170**:891 (Oct.) 1933.

Three young brothers who had no accommodation and were able to read only with a +4 diopter convex lens were observed. They were not hyperopic, and therefore vision for distance was diminished by convex lenses of +0.75 diopter. There was no family history of syphilis or any history of diphtheria or diabetes. Neurologic examination gave negative results. A sister was normal. All the ocular functions were normal except for accommodation and the reflex to convergence and accommodation, which were absent. Atropine and physostigmine (eserine) had no effect on accommodation. As the motility of the iris was normal, the failure of accommodation seemed to be due to aplasia of the ciliary muscle rather than to partial congenital paralysis of the third nerve.

BERENS, New York. [ARCH. NEUROL. & PSYCHIAT.]

ALMOST COMPLETE ABSENCE OF THE MESODERMAL LAYER OF THE IRIS IN TWO GENERATIONS. OCULAR HYPERTENSION AND POLYCORIA IN ONE CASE. R. ROSSANO, Bull. Soc. d'opht. de Paris, January 1934, p. 8.

In the two cases presented, there existed a malformation of the cornea called "embryotoxin" by certain authors; the iris was reduced to its ectodermal layer covered by a bed of transparent stroma, containing most probably the dilator of the iris. The sphincter of the pupil was markedly visible, and the function was normal. In the first case, that of the father, there were two complete lacunae of the iris in the right eye, while the left eye showed hypertension. In the second case there were no lacunae and no hypertension, but the malformations of the corneas and irides were similar to those in the other case. Accompanying the article are photographs of the irides, detailed reports of the patients, an adequate discussion of the literature and bibliography.

MAYER, Chicago. [ARCH. OPHTH.]

A CASE OF CONGENITAL MEMBRANOUS ANKYLOBLEPHARON. C. CORDERO, Arch. di ottal. **40**:369 (Sept.-Oct.) 1933.

In an infant of 39 days the middle portions of the upper and lower lids, over an area 4 mm. long, were connected by a thin membrane. No cilia were present on either lid. The membrane was excised, and sections showed it to consist of epithelium and vascular connective tissue containing muscle fibers and numerous subepithelial glands. The opening of the palpebral fissure in new-born kittens was investigated histologically. The process begins with the formation of spaces filled with keratinized epithelium, usually surrounding a cilium. This keratinization of the epithelium begins at each end of the future palpebral fissure and extends toward the middle. As the cause of the anomaly in his case Cordero supposes an arrest of development of the epithelium and a growth of mesodermal tissue between the borders of the lid to form the membrane observed.

GIFFORD, Chicago. [ARCH. OPHTH.]

ABNORMAL ANLAGE OF THE CILIARY BODY IN A MICROPHTHALMIC EYE. F. FISCHER, Arch. f. Ophth. **132**:71 (March) 1934.

One of the two microphthalmic eyes removed from a child at autopsy showed the following unusual changes: The globe had a sagittal diameter of 5 mm. The cornea, anterior chamber, iris, pupil, ciliary body and lens were absent. The

entrance of the optic nerve was very wide and filled up with glial and mesodermal tissue and nerve fibers. The optic nerve itself was very thin. Around its entrance into the globe lay an extensive coloboma. There the pigmented epithelium was absent, and the choroid was thin and carried only very few vessels. The retina was present as a double but severely changed layer. Outside of the colobomatous area the choroid and pigmented epithelium were normal. The coloboma was bordered on its lateral side by an anlage of the ciliary body located in the posterior segment, which contained numerous well formed processes. Fischer discusses the probable mechanism of this unusual malformation.

KRONFELD, Peiping, China. [ARCH. OPHTH.]

MACULOCEREBRAL TYPE OF HEREDODEGENERATION OF THE MACULA LUTEA. R. BRAUN, Klin. Monatsbl. f. Augenh. **91**:182 (Aug.) 1933.

After excluding Tay-Sachs and Vogt-Spielmeyer-Stock diseases and also syphilitic chorioretinitis and retinitis pigmentosa, the author finds that genuine degeneration of the macula lutea may or may not be accompanied by symptoms of involvement of the central nervous system. These symptoms may be so slight in some cases that only an exact neurologic examination may reveal them. He reports a case of this description. A healthy young woman, aged 23, had had faulty vision since childhood. Vision decreased for about nine months prior to admission to the hospital, when it was 1/8 of normal in the right eye and 6/60 in the left. Central scotomas for colors and slight eccentric fixation were present in both eyes. Bilateral round foci the size of the disk were seen in the macular region; yellowish dots were noticed within these foci, and pigment was seen in their surroundings. The temporal portion of the disks showed slight pallor. Neurologic examination revealed slight weakness of the right facial nerve, left-handedness and facility in mirror writing with the left hand, with which the patient wrote regular script when a child. No disturbance of speech was noticed when she began to write with the right hand. Her mother and a sister suffered from occasional epileptic attacks. Braun considers this a borderline case of the type described by Behr.

STOLL, Cincinnati. [ARCH. OPHTH.]

VARIATION IN THE HORIZONTAL AND VERTICAL DIAMETERS OF THE CORNEA AND IN THE SURFACE AREAS OF THE CORNEA AND SCLERA IN MEGALOCORNEA, NORMAL CORNEA AND MICROCORNEA. R. FRIEDE, Ztschr. f. Augenh. **81**:132 (Aug.) 1933.

THE SURFACE AREAS OF THE CORNEA AND SCLERA IN THE EMBRYO AND THE NEW-BORN AND THEIR RELATION TO MEGALOCORNEA IN THE ADULT. R. FRIEDE, Ztschr. f. Augenh. **81**:213 (Sept.) 1933.

The theme of the author in both these articles is that megalocornea is not a form of giant growth associated with enlargement of the eyeball, but that it is an increase in the surface area of the cornea at the expense of the sclera. Normal relations in the surface areas of these two structures may be expressed by the term "normal cornea, normal sclera." Megalocornea may be designated as "macrocornea, microsclera" and microcornea (without microphthalmus) as "microcornea, macrosclera."

In the first article the author presents tables and graphs based on measurements with the Wessely keratometer of the corneal diameters in 10,940 eyes. The length of the horizontal diameter varied between 10 and 13.5 mm., with an average of 11.56 mm. The vertical diameter varied from 9 to 13 mm., with an average of 11.25 mm. The ratio of the corneal surface to that of the sclera is 1 : 4.76. If this ratio is approximated in any eye, one may speak of normal cornea, normal sclera.

In his second article the author exhibits tables showing measurements of the cornea in the human embryo, new-born infant and child up to 5 years of age. Comparisons are made with the horizontal diameter of the eyeball. The ratio of the corneal to the scleral surface area is placed at 1 : 3.624, to be compared with the

adult ratio of 1 : 4.76. Megalocornea, therefore, is the natural form in the embryo and early childhood. A graph based on measurements of corneal and eyeball diameters in an ascending series of animals, human embryos, infants, young children and adults indicates that megalocornea is the natural form in animals and persists in a lessening degree in the human embryo and childhood.

BEGLE, Detroit. [ARCH. OPHTH.]

CONGENITAL ECTOPIA OF THE LENS INHERITED THROUGH FOUR GENERATIONS. H. ROESE, Ztschr. f. Augenh. **81**:149 (Aug.) 1933.

The author states that congenital ectopia of the lens is of infrequent occurrence. Grob reported 18 cases in the Zurich eye clinic among 68,500 patients over a period of thirty-eight years. Ringelhan and Elschnig mentioned 23 cases among 192,000 patients attending the eye clinic at Preha within the last twenty-three years. In almost half of the reported cases there is evidence of inheritance, and the rarity of the deformity is emphasized if the affected families only are counted. The author outlines a genealogical tree showing nine members with the deformity in four generations. Since most of the afflicted persons were observed over a long period, numerous complications appeared, and the author comments on changes in the position of the lens, luxation of the lens into the vitreous or anterior chamber, attacks of increased tension, scleral staphyloma and axial myopia with choroidal degeneration.

BEGLE, Detroit. [ARCH. OPHTH.]

## Diseases of the Ear, Nose and Throat

IODIZED OIL IN BRONCHIECTASIS. JOSEPH A. WEINBERG, Arch. Surg. **27**:545 (Sept.) 1933.

The author makes several deductions as to the fate of iodized oil in bronchography and in the treatment of bronchiectasis. His conclusions are based on a study of two cases following lobectomy and on experiments with iodized oil in animals. Postoperative study of two bronchiectatic human lungs into which injections of iodized oil were made showed that iodized oil may be retained in the lung for at least eighty-three days following its introduction into the bronchial tract and that most of the cells found in the lung are capable of ingesting droplets of the oil.

Experiments with animals showed that an excessive amount of iodized oil was a factor in the development of pneumonitis. This occurrence and the occurrence of atelectasis in these animals indicate that only small amounts of iodized oil should be used for diagnostic purposes in clinical work.

KAISER, Rochester, N. Y.

SPEECH DISORDERS AS A MEDICAL PROBLEM. SMILEY BLANTON, New York State J. Med. **33**:215 (Feb. 15) 1933.

As speech is one of the highest functions of human organization it is also one of the best indicators of the state of development of the central nervous system, especially the brain. Defects in speech due to organic lesions of the brain are seen in multiple sclerosis, dementia paralytica and hemorrhage of the brain. They are also found in myasthenia gravis.

Little's disease is frequently accompanied by slurring or indistinct speech. In such cases training in speech should be in small doses and should be carried out over some time. Such training should be supervised by the physician to avoid overtreatment. The child who does not talk at the age of 2 or 2½ years may have a defect. The most common cause of delayed speech in children is amentia due to lack of brain development. Minute hemorrhages at birth or later encephalitis may be the cause. The attention of such children is too fixed. The normal child does not fix his attention on one thing for a long time. The correction of defects

in speech due to deafness requires teachers experienced in the training of the deaf.

Sometimes indistinct and slurring speech is found in bright children, owing to oral inactivity. Severe illness between the fifteenth and twenty-fourth months may cause retardation in speech. Tonguetie is rarely the cause. Defects in speech are often used by children as a means of dominating parents and nurses. The substitution of "th" for "s" may be due to the desire to remain an infant. Children who show this tendency should not be given things that they desire until they say the words clearly and distinctly.

Stuttering is caused by emotional factors, such as timidity or anxiety. From 5 to 10 per cent of nursery children stutter for a period; 2 or 3 per cent stutter for several months, and 1 per cent of the general population stutter. Exercises in speech should not be used for young stuttering children. Better results are obtained by a change of environment. Remove pressure, demand less response in speech and give the child more freedom. Teach the child to adjust himself to situations through games, plays and mental hygiene. Build up a feeling of confidence and security. By modification of personality tendencies, such as marked anxiety, sensitiveness or overaggressiveness, defects in speech can be eliminated.

AIKMAN, Rochester, N. Y.

LUNG ABSCESS AFTER TONSILLECTOMY. F. T. RANSON and L. MCGOLRICK, Brit. M. J. **2**:1020 (Dec. 2) 1933.

The authors report a case of a pulmonary abscess following tonsillectomy, with operation and recovery. A brief discussion of the mode of production of abscesses and the treatment follows.

ROYSTER, University, Va.

THE "ACUTE EAR" IN GENERAL PRACTICE. DOUGLAS GUTHRIE, Brit. M. J. **1**:4 (Jan. 6) 1934.

The author considers the causes and clinical course of acute otitis media, the atypical varieties of this condition, the appearance of the drum, the appearance of the patient, the indications for incision and the complications, including mastoiditis, along more or less well known and conventional lines.

ROYSTER, University, Va.

HAEMATURIA IN CHILDHOOD ASSOCIATED WITH ACUTE MASTOIDITIS. LAURA L. BATEMAN, Brit. M. J. **1**:60 (Jan. 13) 1934.

A case of acute mastoiditis accompanied by hematuria in a child 6 years of age, is reported. An operation was performed on the mastoid, and within five days the hematuria had disappeared. The conclusion is that hematuria associated with mastoiditis indicates early operation.

ROYSTER, University, Va.

SARCOMA OF THE NOSE IN AN INFANT. HAROLD EDWARDS, Proc. Roy. Soc. Med. **27**:27 (Nov.) 1933.

A case of sarcoma of the nose was reported in a child aged 2 years. The patient had been cured when a few months old by treatment with radium, goat serum and erysipelatous and prodigiosus toxins (Coley's fluid). Eight months later a lump appeared in the submaxillary region; the subsequent history is given in the report.

WILLIAMSON, New Orleans.

ADENOIDITIS, ADENOPATHY AND NASOPHARYNGEAL MASSAGE. G. VERGER, Arch. de méd. d. enf. **37**:21 (Jan.) 1934.

Forcible friction of the adenoid area by the finger was first practiced in 1926 by Robert Rendu of Lyon for inflammation of adenoid vegetation accompanied by regional adenopathy. In 1929, Gamaleia and Michon used the term "providential

finger" in discussing the agent used in crushing these tissues. Although this method was first employed only in cases of prolonged adenoiditis, Verger now uses it in the acute stage, with equal success; the elevation in temperature, which may continue for many days in untreated patients, usually falls to normal on the day following massage. Verger believes that whether or not adenopathy accompanies acute adenoiditis, it is the latter invasion which is responsible for the rise of temperature, or what is commonly known as glandular fever.

It is maintained that acute prolonged adenoiditis probably constitutes the chief source of continued hyperthermia in early childhood. The author reports seventeen cases observed, including cases of simple acute adenoiditis, several of the prolonged type and a number of instances in which cervical adenitis was a complication.

Before crushing the vegetation it is necessary to exclude otitis media; if this exists and is properly treated without causing a drop in the temperature, adenoiditis is suspected and is treated as has been described. Tuberculosis is excluded by a negative cutireaction and consideration of the child's environment; the elevation in temperature in the latter disease is also much more regular in its stride. Central pneumonia is difficult to differentiate at this time. It is assumed that the favorable action noted in this procedure follows the escape of minute quantities of pus collected between the lamellae of the adenoids.

AMESSE, Denver.

THE TONSIL PROBLEM. G. M. SUSIN, Prat. pediat. **11**:323 (Oct.) 1933.

Susin summarizes the various theories concerning the function of the tonsils and discusses the disparity in reported results of tonsillectomy. He concludes that a conservative attitude should be taken toward tonsillectomy and that even in cases of acute and chronic tonsillitis partial removal is preferable to complete extirpation. A good bibliography of the Italian literature on this subject is listed.

DOOLEY, Kent, Conn.

THE CAUSES OF DEAF-MUTISM. SUNÉ y MEDAN, Paidoterapia **12**:1099 (March) 1934.

The author gives special attention to the latent otitis of the sucking baby. He insists above all that hereditary deaf-mutism is as frequent as acquired deaf-mutism, and Parrel admits that the number of persons with congenital deafness is greater than the number of those with acquired deafness.

Syphilis is an important cause of this condition. It may produce deafness before or after birth, at about the fifth year or, if late, from the seventeenth to the thirtieth years. If it supervenes before the child starts talking, he will remain a deaf-mute. The Wassermann reaction has only a relative informative value. Other congenital causes include a hereditary tendency, physical traumatism of the mother during the pregnancy, nervous defects and consanguinity. Deafness due to consanguinity is as high as 10 per cent, according to some authors.

Causes of acquired deafness include traumatism to the head during early infancy, cerebrospinal meningitis and latent otitis media of the suckling. Latent otitis occurs most frequently from the sixth to the ninth month. It is best prevented by careful examination of the ears of all infants and the early treatment of otitis; by the prevention of marriages between blood relatives, especially when there is deafness in the family of one of the parties, and by repeated careful examination of the ears of all infants with fever.

HERRON, Spokane, Wash.

EXPERIENCES WITH INTUBATION. MARTIN HOHLFELD, Jahrb. f. Kinderh. **138**:334 (March) 1933.

Dorner published a monograph dealing with tracheotomy (Die Diphtherie-Epidemie in Leipzig in den Kriegsjahren 1914-16 nach Beobachtungen im Krankenhaus St-Jakob, Jena, Gustav Fischer, 1918). Recently Hohlfeld published an

extensive analysis of his experiences at the Kinderkrankenhaus in the same city during the same period, in which he shows that intubation gave about from 11 to 12 per cent better results, even in cases complicated by measles.

CLAUSEN, Rochester, N. Y.

OSTEOMA OF THE FRONTAL SINUS. E. KNAPP, Monatschr. f. Ohrenh. **67**:903 (Aug.) 1933.

Knapp reports a case of osteoma of the frontal sinus in a girl, aged 15. The clinical symptoms were a visible flat protuberance above the root of the nose and adherent to the bone, a small palpable protuberance on the inner canthus and next to it on the orbital margin a small pulsating tumor. The tumor of the frontal sinus was diagnosed roentgenologically. At operation it was seen that the osteoma had broken through the anterior wall of the frontal sinus. The pulsating tumor was a mucocele. The osteoma had also perforated the central and inferior parts of the posterior wall of the sinus and had become adherent to the dura. In an attempt to isolate the tumor at this point, the dura was ruptured. The operation was terminated without removal of the tumor. Meningitis did not result.

EDITOR'S ABSTRACT. [ARCH. OTOLARYNG.]

TRAUMA SUSTAINED AT BIRTH AND ITS INFLUENCE ON THE ORGAN OF HEARING. H. BARTH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:1 (Nov. 10) 1933.

Barth studied the influence of trauma sustained at birth on the organ of hearing from the standpoint of the otologist. His investigations included: histologic examination of many temporal bones to determine the frequency and the significance of hemorrhages; clinical examination of many nurslings to ascertain whether spontaneous nystagmus may be considered as a sign of birth trauma, and examination of the hearing of ninety-six children between the ages of 7 and 12 years who had been delivered by forceps. He concludes that one has to be cautious in interpreting histologic pictures as well as in drawing conclusions as to later life on the basis of them. He did not find a single case in which deafness or baryecoia verging on deafness resulted from an abnormal delivery. He does not agree with the view that spontaneous nystagmus is a sure sign of birth trauma; it is probably a physiologic phenomenon. While birth trauma must be considered in the problem of the etiology of deafness, he thinks that it does not play nearly so important a rôle as has been ascribed to it by some investigators.

EDITOR'S ABSTRACT. [ARCH. OTOLARYNG.]

SEPSIS FOLLOWING ANGINA AND TONSILLECTOMY. JAN HYBASEK, Časop. lék. česk. **71**:993 (Aug. 5); 1034 (Aug. 12); 1078 (Aug. 19) 1932.

The author discusses the various types of tonsillitis from the clinical, etiologic, symptomatic and diagnostic aspect and cites the contemporary literature. He classifies tonsillitis as follicular, phlegmonous, pseudomembranous and vesicular. The therapeutic measures used are outlined. A series of reports of cases is presented, and the prognosis is discussed. The résumé of active treatment is as follows: Drainage in acute sepsis is more important than tonsillectomy. The indications for tonsillectomy should be limited rather than amplified in postanginal sepsis. Peritonsillar abscesses should be opened widely orally, and tonsillectomy should not be performed in the presence of a peritonsillar phlegmon. Prophylactic jugular ligation should not be done, but thrombosed vessels should be ligated or excised. The deep structures of the neck and the parapharyngeal regions should be opened widely and drained. A bibliography is appended.

STULIK, Chicago.

CENTRAL DISORDERS OF HEARING IN ABSCESSES OF THE TEMPORAL LOBE. L. M. KOMPANEYTS, Vrach. delo **16**:383, 1933.

The author reports five cases of abscesses of the temporal lobe, and gives the results of the investigation of the auditory changes. The observations presented are the result of studies of the auditory changes on the side opposite that on which the lesion was located, since it is known that most fibers of the auditory nerve decussate and because too many complicating factors are present on the same side. The results obtained were: (1) shortening of the bone conduction with the low tone tuning-forks C and A and (2) diminished perception of sound for the lower range tones, i. e., either by air or bone conduction.

The author's conclusions from the results obtained are:

1. For normal qualitative perception of sound, the integrity of both auditory and subcortical centers is necessary.

2. With the involvement of the auditory center and subcortical tracts on one side, there is observed a diminution in the perception of sound, particularly for the lower range of the auditory scale. This would suggest that the decussated fibers of the auditory nerve are primarily fibers for the conduction of the low pitched tones, i. e., that there is a differentiated mechanism for the perception of sound.

3. The shortened bone conduction in abscesses of the temporal lobe would tend to show that the perception of sound through bone conduction depends not only on the condition of the peripheral nerve endings but on the cerebral cortex as well.

BODER, Chicago.

## Diseases of the Skin

NEVUS FLAMMEUS NUCHAE: ITS OCCURRENCE AND ABNORMALITIES. EDWARD F. CORSON, Am. J. M. Sc. **187**:121 (Jan.) 1934.

Nuchal nevi are described as one of the most common congenital defects of the skin and are a variety of flat angiomas. They are located usually on either side of the posterior midline of the neck and are fairly symmetrical, with a long axis up and down and lacy, jutting, broken up, indented and irregular edges. They are not elevated and vary in color from light pink to purplish red.

According to statistics based on a study of seven hundred and sixty cases, nuchal nevi seem to occur in approximately 5 per cent of the population and are found more frequently in adults (5.6 per cent) than in the new-born (2.8 per cent.). Both sexes are about equally affected. The usual inflammatory change on this abnormal skin is in the nature of a dermatitis, seborrheic or eczematoid. There is an occasional resemblance to psoriasis. The nevus serves as a starting point for outbreaks, mainly in long-haired persons. Eight cases are reported, and one picture of a typical nuchal nevus is shown.

HENSKE, Omaha.

JUVENILE ELASTOMA. FRED D. WEIDMAN, NELSON PAUL ANDERSON and SAMUEL AYRES JR., Arch. Dermat. & Syph. **28**:182 (Aug.) 1933.

The authors report a case of congenital abnormality of the elastic tissue to which they give the name "juvenile elastoma." This occurred in a white child, 5½ years old. Over the trunk, buttocks and lateral and anterior aspects of the thigh appeared numerous discrete, pea-sized, flesh-colored to slightly yellow, doughy, rather flat nodules. Histologic examination of a nodule revealed the elastic tissue to be in a degenerating stage, less wavy than normal and extending below the sweat glands. Care has been taken to differentiate it from other types of degenerative diseases seen in elastic tissue. At present the case does not resemble pseudoxanthoma elasticum, but the possibility of its being indistinguishable from the latter condition by the time the child has reached adult life was not overlooked.

DULIN, Iowa City.

CLINICAL VARIANTS OF FAMILIAR DISEASES OF THE SKIN: PHENOLPHTHALEIN ERUPTION; PITYRIASIS ROSEA; LICHEN PLANUS; PARAPSORIASIS. LEONARD F. WEBER and HERBERT RATTNER, Arch. Dermat. & Syph. **28**:190 (Aug.) 1933.

The authors report rare types of these diseases, as follows: The first case was one of large bullous eruptions of the palms following the ingestion of phenolphthalein. The second case was one of pityriasis rosea with the usual type of lesion on the body, extremities and neck; on the feet were numerous closely arranged, deep-seated vesicular lesions on inflamed bases, which were similar to those that occur in vesicular ringworm infection. The third case was one of lichen planus limited to the soles. The fourth case was one of lichenoid parapsoriasis in which the eruption was bilateral and symmetrical and involved the trunk, extremities and the palms and soles. Because of its resemblance to syphilis, this patient was at one time given a course of antisyphilitic treatment. Since then the author has observed her at intervals for three years, and the lesions have remained unchanged. It is rare to find the palms and soles involved in this condition.

DULIN, Iowa City.

SUCCESSFUL TREATMENT OF VITILIGO. MILTON H. COHEN, Arch. Dermat. & Syph. **28**:215 (Aug.) 1933.

The author gives considerable hope in cases of vitiligo when the lesions are treated with a 10 per cent alcoholic solution of oil of bergamot twice daily, followed by semiweekly exposure to ultraviolet rays for periods of from three to five minutes. A weekly intravenous injection of gold sodium thiosulphate (0.1 Gm.) is given. Cohen had a patient treated in this manner to whom fourteen injections of gold sodium thiosulphate were given along with the other treatment. The pigment had entirely returned to areas on the face and arms, and the patient had had no return of the lesions after a year's observation.

DULIN, Iowa City.

INCIDENCE AND PROPHYLAXIS OF EPIDERMOPHYTOSIS IN SCHOOL CHILDREN. LOREN W. SHAFFER and W. R. CAREY JR., J. Michigan M. Soc. **32**:648 (Dec.) 1933.

The authors set out to investigate the incidence of epidermophytosis in the public schools of Detroit, its mode of transmission and the effect of prophylactic measures. They are of the opinion that both the incidence and severity of the infection increase at puberty, owing to increasing susceptibility rather than to exposure. The social status of the child appeared to be of little influence in the spread or incidence, yet there seemed to be a familial tendency toward the infection. Sodium hypochloride appears to be the most promising agent for both prophylaxis and treatment.

CAREY, Detroit.

A NOTE ON THE USE OF LASSAR'S PASTE IN THE "WEEPING ECZEMA" OF INFANCY. PARK J. WHITE, J. Pediat. **4**:104 (Jan.) 1934.

An infant with weeping eczema of the face was treated with plain strong or mild resorcinol paste. A few days later he was brought back to the clinic with a temperature of 104 F. There were a mucopurulent discharge from the nose and thick, firm crusts all over the face, at the outer edges of which were undermined circinate blebs typical of acute spreading impetigo. Several points of practical importance are cited from this case. In the local treatment of acute moist eczema the likelihood of secondary infection must be kept in mind. Crusts should be removed with oil before pastes are applied. Too great a proportion of powder should not be incorporated in the paste. A mild disinfectant-stimulant, such as crude oil tar, should be more generally added. Applications and dressings should be changed frequently.

LEMASTER, Evanston, Ill.

ERYTHEMA NODOSUM. W. R. F. COLLIS, Brit. M. J. **2**:1162 (Dec. 23) 1933.

The author reviews briefly the evidence in favor of the rheumatic, tuberculous and acute specific origin of erythema nodosum and brings out his views to the effect that erythema nodosum is an allergic manifestation and may be brought about by more than one type of allergy. He is of the opinion that by far the commonest cause is tuberculous infection. The streptococcic type is extremely rare. He thinks that every case of erythema nodosum should be considered tuberculous until proved otherwise.

ROYSTER, University, Va.

SWIFT'S OR PINK DISEASE: RECENT WORK ON PATHOLOGY AND TREATMENT. S. F. MCDONALD, M. J. Australia **2**:276 (Aug. 26) 1933.

The observations of Greenfield and Paterson in regard to this disease are cited as showing diffuse infiltration with round cells, which is most prominent in the cervical and lumbar enlargements and never takes place above the medulla. There are chromatolytic changes of an axonal type in these areas, which are considered as being secondary to degeneration of the peripheral nerves. Some changes were also noted in the sympathetic and parasympathetic nerves.

The theories of etiology are briefly reviewed, and the work of Wyllie and Stern is cited as giving evidence of a nutritional disorder. These investigators observed a condition similar to Swift's disease in rats when the only source of protein was dried egg-white. The condition was promptly cured by adding liver extract to the diet.

The author reviews several cases in which liver extract was used with apparent success. However, he thinks that the clinical control group was too limited and the observations on these cases were not close enough to be more than suggestive.

TENNEY, Madison, Wis.

RAYNAUD'S SYNDROME TREATED BY SYMPATHECTOMY. A. V. NEALE and F. A. R. STAMMERS, Proc. Roy. Soc. Med. **26**:1542 (Oct.) 1933.

A girl, aged 4 years, was found on admission to the hospital to be suffering from a definite intermittent Raynaud syndrome of the hands and feet, associated with a certain amount of scleroderma. Bilateral lumbar sympathectomy was performed. It was proposed to await the results of the operation; if they were satisfactory, cervical sympathectomy would be carried out.

WILLIAMSON, New Orleans.

MULTIPLE SUBCUTANEOUS NEOPLASTIC TUMORS IN AN INFANT OF FOUR MONTHS. L. MORQUIO, Arch. de méd. d. enf. **37**:29 (Jan.) 1934.

A case difficult to interpret is presented from the pediatric service of the University of Montevideo. An infant aged 4 months was admitted from a country district; it was the first child of healthy parents with no history of consanguinity or miscarriage; it had been born at full term and was breast fed. Fifteen days after birth the mother noted the appearance of small indurated nodules scattered over the limbs and trunk; several of these later became soft and finally suppurated. Examination on the fortieth day of life revealed a strong, vigorous infant with a small subcutaneous abscess and multiple indurated nodes of unknown etiology. Tumors continued to appear, especially about the head. At the age of 4 months these became hemorrhagic; they varied in size from that of a grain of corn to that of a hickory nut and were scattered over the entire body; they were adherent to the skin and resembled fibrous tissue in consistency.

In addition to this eruption, the fontanel was unusually large, and craniotabes was found. Both the liver and the spleen were moderately enlarged. The Wassermann reaction of the blood both of the mother and the child was negative, as was also the cutireaction for tuberculosis in the patient. The blood picture showed

nothing abnormal, and roentgenograms were essentially normal. Biopsy was performed on a nodule removed from the leg, and the existence of a cellular infiltration characteristic of neoplastic tissue was demonstrated. Some days later, after the baby had returned home, a generalized eruption suddenly developed, with elevation of temperature and prostration. The patient died on the third day of the invasion, the diagnosis being scarlet fever (with a question mark). There was no autopsy. The author speculates on the differentiation between reticulo-endotheliosis, diffuse sarcomatosis and the even rarer tumors, sympathicoblastomas.

AMESSE, Denver.

SEASONAL FREQUENCY OF SOME SKIN DISEASES IN INFANCY AND CHILDHOOD. HENRY KAMMER, Arch. f. Kinderh. **101**:17 (Dec.) 1933.

A survey of the diseases of the skin in children seen at the University Clinic at Freiburg during the past nine years showed that eczema in children over 3 months of age occurred predominantly during the months from December to April. This seasonal occurrence is striking. On the other hand, eczema in infants under 3 months of age and erythrodermia desquamativa of Leiner showed an irregular frequency curve throughout the year. The author believes that these findings indicate that eczema in children under 3 months of age and Leiner's disease are probably entirely separate from the true eczema that occurs in older infants and children.

LEMASTER, Evanston, Ill.

A FAMILY EPIDEMIC OF ERYTHEMA NODOSUM. P. L. ROTNES, Dermat. Ztschr. **67**:259 (Sept.) 1933.

In four sisters living in the same house, two adults and two children, erythema nodosum developed within five weeks. The tuberculin reaction was strongly positive in all the patients, and roentgen examination disclosed positive findings for tuberculosis in the two younger sisters. The author concludes that (1) the erythema was the same in all four patients; (2) the tuberculous infection was a condition *sine qua non* for its development; (3) the location, appearance and course of the eruption could not be differentiated from those of idiopathic erythema nodosum; (4) erythema nodosum may occur on the basis of a tuberculous process in adults as well as in children, but whether this is always the case is not proved.

TAUSSIG, San Francisco. [ARCH. DERMAT. & SYPH.]

PERIPHERAL BLOOD CHLORIDE IN EXUDATIVE DIATHESIS. G. TOROK, Monatschr. f. Kinderh. **59**:190, 1934.

A study of the chlorides of the peripheral blood in exudative diathesis shows that in the normally nourished infant, exudative diathesis causes no increased chloride content. In the infant with exudative diathesis who is overweight and who has a pasty appearance, chlorides may be increased. In the malnourished infant with this condition there may also be an increased chloride, but this increase is also found in malnourished infants not suffering from the diathesis.

GERSTLEY, Chicago.

A PREVIOUSLY UNDESCRIBED ANOMALY OF PIGMENTATION: DYSCHROMATOSIS UNIVERSALIS HEREDITARIA. IKUZŌ TŌYAMA, TOKUJI ICHIKAWA and YOSHIO HIRAGA, Jap. J. Dermat. & Urol. **34**:360 (Oct.) 1933.

Except for the greater extent, this anomalous condition of the skin resembles the condition described as acropigmentatio by Komaya in 1920, as leukopathia punctata et reticularis by Matsumoto in 1927 and as dyschromatosis symmetrica hereditaria by Tŏyama four years ago. In the present cases the condition, which involved the entire bodies of the patients, with the exception of the palms and

soles, consisted of pigmentary flecks, varying in shape and size from those of a grain of rice to those of a bean, but being usually about the size of a pea. The lesions were sharply delimited and thickly set, and the intervening areas of leukoplakia were of like shapes and sizes. The condition was not so pronounced in the younger patients, and different stages of the process could be observed in the group. The hereditary character of the condition was indicated by the fact that in one family the condition was present in the mother and in both children (males) and that in the other it occurred in the mother and in two (females) of the six children, who were of both sexes. BRENNAN, Chicago. [ARCH. DERMAT. & SYPH.]

## Surgery and Orthopedics

THE RESULTS OF SYMPATHECTOMY IN THE TREATMENT OF PERIPHERAL VASCULAR DISEASES, HIRSCHSPRUNG'S DISEASE AND CORD BLADDER. ALFRED W. ADSON, Ann. Int. Med. **6**:1044 (Feb.) 1933.

The author reports eight cases of Hirschsprung's Disease in which sympathectomy was performed without a fatality. The patients were symptomatically relieved, but the colons did not return to normal size following operation.

READING, Galveston, Texas.

UNILATERAL PYONEPHROSIS IN EARLY INFANCY. IVING J. SHAPIRO and ARTHUR F. ABT, Arch. Pediat. **50**:615 (Sept.) 1933.

Attention is called to the rarity of unilateral lesions of congenital origin in the upper urinary tract. Two cases of this nature are reported in which nephroureterectomy was successfully performed. WADDELL JR., University, Va.

OSTEOMYELITIS OF THE SKULL. ABRAHAM O. WILENSKY, Arch. Surg. **27**:83 (July) 1933.

Acute osteomyelitis of the skull caused by the common pyogenic organisms is thoroughly discussed. It is an infrequent occurrence. One half of the cases reported occurred in children and adolescents. The infection arises as a primary or extension process or occurs as a manifestation of a hematogenous infection. It is rare as a primary disease, and is most common as an extension process after nasal accessory sinus disease; it is relatively rare as a hematogenous infection, and it is least common as a complication of an otologic infection.

The organisms invade the diploe of the bone, and osteomyelitis results. The infection either spreads through the outer table and produces subperiosteal abscesses beneath the scalp or spreads inward to give rise to extradural or subdural abscess, general meningitis, cerebral abscess or thrombosis of the longitudinal sinus.

Extensive operation is necessary when the diagnosis of osteomyelitis is evident. The mortality in the author's series, as well as in other series discussed, was about 50 per cent.

KAISER, Rochester, N. Y.

A FOREIGN BODY IN THE VAGINA OF A FOUR YEAR OLD CHILD, WITH RESULTANT MASSIVE HEMORRHAGE. S. NIXON, J. A. M. A. **100**:1169 (April 14) 1933.

A safety pin was removed from the vagina of a child, aged 4 years, who had had multiple vaginal hemorrhages. The pin was buried in the vaginal and interstitial portion of the cervix and the parametrial structures and had caused necrosis of the surrounding tissues.

BONAR, Salt Lake City.

CONGENITAL HYPERTROPHIC PYLORIC STENOSIS. TREATMENT OF ACCIDENTAL PERFORATION OF MUCOSA DURING RAMSTEDT OPERATION. O. M. LAMSON, Surg., Gynec. & Obst. **57**:398 (Sept.) 1933.

The theory that hyperplasia of the pylorus is the result of spasm alone does not explain extreme cases in very young infants without acceptance of a congenital origin. Pyloric hypertrophy is about seven times as frequent in males as in females and occurs in about one of every two hundred male infants born. Its occurrence two or three times in one family has been reported.

Since the demonstration of Ramstedt's operation in 1912 it has generally superseded other surgical procedures as the treatment involving least hazard for patients with extreme stenosis. It carries the risk, however, of accidental perforation of the mucosa in spite of due precaution. On experiencing this misfortune in operating in a recent case Lamson successfully devised a method of plastic repair for accidental perforation. The method makes use of an oblique incision extending through the muscular coats at an acute angle away from the central portion of the main longitudinal incision in such manner as to secure a triangular flap suitable for suturing to the thickened muscle coats over the site of the perforation. This method, while particularly applicable to the repair of a perforation at the usual site (i. e., the distal end of the incision in the duodenum) may be used with appropriate modification for the repair of a perforation in the midportion of the mucosa. Diagrams illustrate the technic, which has been found superior to the customary purse string suture and omental transplantation.

GRAVES, New York.

THE SURGICAL TREATMENT OF BRONCHIECTASIS. ROBERT M. JANES, Brit. J. Surg. **21**:257 (Oct.) 1933.

Since the introduction of iodized poppy seed oil 40 per cent by Forrestier in 1922 the surgical treatment of bronchiectasis has advanced considerably. At present it may be divided into three main groups: (1) collapse therapy as obtained by pneumothorax, section or exeresis of the phrenic nerve or thoracoplasty; (2) pneumotomy with drainage of the larger collections of pus, and (3) excision or destruction of the involved pulmonary tissue.

From the pathologic picture produced by the disease it seems obvious that alleviation of symptoms is all that can be hoped for from collapse therapy. Results obtained by various workers are quoted to verify this observation. Pneumothorax is recommended as a preoperative method to aid in emptying the dilated bronchi of their infective contents. The results of extrapleural thoracoplasty indicate that it should be reserved for those cases of unilateral bronchiectasis in which the area of lung involved is too great to be removed completely. The operation should be done in four stages.

The results of pneumotomy with drainage of large abscess pockets are about the same as those from collapse. There may be relief from symptoms but never a cure.

Lobectomy has been recognized by most writers as the ideal method of treatment. In this article a complete detailed account of the operation is given, with figures demonstrating each step. The operation was performed by the one-stage method on sixteen patients. Of these, six were cured; three, improved; two, unimproved, and five died—three of bronchopneumonia, one of secondary hemorrhage, and one of abscess of the brain fifty-two days after the operation. The operation is justifiable in patients with a large amount of foul sputum, in those who have suffered numerous pulmonary hemorrhages, and in those in whom the disease appears to be definitely progressive. Patients with persistent fever and those who have recovered from exacerbations of the disease are unsuitable for the operation. It is advisable to allow a considerable afebrile interval to elapse before doing a lobectomy.

SUMMERFELDT, Toronto, Canada.

PULMONARY LOBECTOMY. J. E. H. ROBERTS and H. P. NELSON, Brit. J. Surg. **21**:277 (Oct.) 1933.

This article gives reports of cases and the results of surgical intervention in ten cases of lobectomy. Except for a few minor changes the operative technic was similar to that of Shenstone and Janes. Preoperative, operative and postoperative treatment and postoperative complications are discussed. The results obtained were fairly satisfactory. In six of the ten cases healing took place and no symptoms remained. Two patients were at work, but had slight symptoms. One patient died ten days after the operation from a cerebral abscess, and one died of influenzal pneumonia three months after the operation. The ages in this group varied from 13 to 55 years. Roberts and Nelson conclude that the mortality from lobectomy is considerably reduced by the use of this method and that this operation will take its place as the treatment of choice in cases of unilobular bronchiectasis.

SUMMERFELDT, Toronto, Canada.

TUBERCULOSIS OF THE HIP JOINT. GERTRUDE HERZFELD, Tr. Edinburgh Med.-Chir. Soc., 1933, p. 171; in Edinburgh M. J., September 1933.

In the case of a 5 year old boy with tuberculosis of the hip joint, the operation of fusing the joint was that of Wilson, which consists in turning down a pedicled flap from the ilium and inserting it into the split trochanter and upper part of the femur. The graft stayed in position, but in such cases there is a tendency toward fracture of the grafted portion. Continued immobilization should permit the graft to become stabilized.

NEFF, Kansas City, Mo.

A NEW TECHNIC IN THE ANESTHESIA OF THE BRACHIAL PLEXUS AND ITS ADVANTAGES IN ORTHOPEDIC SURGERY. M. MARTINEZ CURBELO, Cir. ortop. y traumatol. **1**:87 (April) 1933.

The author describes a new technic (a variation of Kulenkampff's method) for the anesthetization of the brachial plexus; this method eliminates the possibility of accidents. Procaine hydrochloride is the anesthetic of choice, being used after the patient has received morphine and atropine preoperatively. Complete sensory anesthesia is produced and usually complete motor anesthesia as well. The anesthesia works faster and gives surer results in children than in adults. The author considers local anesthesia of the brachial plexus the method of choice when operative procedures are carried out on the upper extremity.

HERRON, Spokane, Wash.

SPINAL ANESTHESIA IN INFANTILE SURGERY. RICARDO DE LA FLOR, Cir. ortoped. y traumatol. **1**:107 (April) 1933.

The author suggests that spinal anesthesia be used more in infantile surgery, particularly in infra-umbilical surgery and in orthopedic surgery of the lower extremities. He insists on the advantages of this anesthesia over ether or chloroform. Gas anesthesia is not mentioned. In twenty cases good results were obtained. He recommends its use in children over 7 or 8 years of age. Proper preparation of the patient by careful physical examination, with close attention to the cardiovascular system, is essential.

Morphine and atropine are used preoperatively. The sitting position is used in performing the puncture, and the lying position (Trendelenburg) during the operation. Procaine hydrochloride is the anesthetic of choice.

HERRON, Spokane, Wash.

THE TREATMENT OF FRACTURE OF THE CLAVICLE IN INFANCY. ALBERTO B. INCLÁN, Cir. ortoped. y traumatol., Habana **1**:231 (Oct.) 1933.

Inclán advocates a thorough roentgenologic examination of the fracture as a preliminary to treatment. His method then is to use resorptive sutures after previous perforation of the fragments. This gives a good union without the danger of infections or pseudarthrosis. The procedure takes from five to eight minutes. He uses a modified Sayre apparatus, which is maintained in place by a plaster cast throughout the period of treatment, thus preventing the fragments from moving. Complications from improper treatment of a fractured clavicle entail a much more unfavorable prognosis than writers on this subject would lead one to believe. There has been no experience with this form of treatment in obstetric fracture, but Inclán believes that it could be used to advantage in such cases.

SANFORD, Chicago.

VULVOVAGINITIS IN INFANCY. CLOVIS C. DA COSTA, Rev. de gynec. e d'obst. **27**:425 (Nov.) 1933.

This condition is rather frequent in Brazil. Da Costa observed 230 cases in 6,000 children admitted to the dispensary of the Hospital da Fundacao Gaffree-Guinle. Of these, 115 showed gram-positive, 69 gram-negative and 47 mixed organisms. In other words, 49.5 per cent of the inflammations were due to gram-positive organisms and the remainder could not be classed as such. Of the 115 patients with gram-positive organisms, 42 per cent discontinued treatment before any results could be obtained, and of the remainder, 33 per cent did not attend the clinic until the condition was fully cured. In treatment, local applications are preferred to vaccines or general therapy. A silver preparation is used. In 12 patients, after the use of the silver preparation the vagina became permanently clear of the organisms.

SANFORD, Chicago.

THERAPY OF JUVENILE MENORRHAGIA. OTTO REICHELT, Wien. klin. Wchnschr. **46**:81 (Jan. 20) 1933.

Reichelt concludes that every therapeutic procedure used for the treatment of juvenile menorrhagia must avoid mutilating operations, inclusive of roentgen castration. Operative action should be considered only when life is acutely endangered, and in these cases curettage is careful therapy. However, in all cases in which time is not limited and in which the clinical picture, the condition of the blood, etc., do not indicate urgent action, it is essential to begin with hormone therapy, which consists of supplying corpus luteum. Transfusions of moderate amounts of blood may accelerate the result, owing to the quantity of blood infused and also to the styptic action and the stimulus on the blood-forming organs. Aside from these considerations, it is to be understood that proper general treatment should be carried on; strengthening nutrition, sufficient fresh air, moderate activity in sports, regular cool baths and regulation of the bowels must supplement all therapeutic measures.

ABT, Chicago.

RESULTS OF TANNIN TREATMENT OF BURNS IN CHILDREN. MAX LANGER, Wien. klin. Wchnschr. **46**:689 (June 2) 1933.

Langer states that since the introduction of tannin treatment, combined with blood transfusions and intravenous injections of sodium thiosulphate, the mortality due to burns has decreased from 14.9 to 7.7 per cent.

The sequelae of burns do not seem to be affected by the various methods of treatment, though they have become rarer within the past few years.

ABT, Chicago.

THE TREATMENT OF EMPYEMA WITH LARGE DOSES OF RIVANOL AND OPTOCHIN. A. DOSKOCIL and J. PROCHASKA, Časop. lék. česk. **71**:1057 (Aug. 19) 1932.

Ethoxydiamino-akridinchloride in a dilution of 1:10,000 was sufficient to kill most of the streptococci in empyema. A 1 per cent solution of this preparation was injected into the pleural cavity after paracentesis in streptococcic empyema and was followed by lavage. The concentration of the dye is ten times as great in the sediment as in the serum, because of the combination with the precipitated proteins. For this reason lavage is important, because the insoluble protein compounds formed hinder subsequent absorption. The preparation must not be injected into either the lung or the subpleural tissues, because of the danger of necrosis. Of seven children treated with this drug one died of streptococcic sepsis; five recovered completely, and one has retraction of the wall of the chest. Ethoxydiamino-akridinchloride is of no value intravenously in sepsis, because it is rapidly destroyed in the liver and kidney. Ethylhydrocupreine hydrochloride was used similarly in pneumococcic empyema, but in a 0.5 per cent solution in lavage and in amounts of a few cubic centimeters in residual instillation of a 5 per cent solution, but never more than 0.5 Gm. was left as a residuum. The disadvantage is that ethoxydiamino-akridinchloride decreases the leukocytic function, and ethylhydrocuprein hydrochloride in large doses produces a rapid decrease in polymorphonuclears in the secretion. The advantage is that there is an inhibition of growth in the streptococci, in the case of the former, and the pneumococci, in the case of ethylhydrocupreine hydrochloride, without absorption of toxic by-products in the pleural cavity.

STULIK, Chicago.

TRAUMATIC RUPTURE OF THE SPLEEN. J. GOUDSMIT, Nederl. tijdschr. v. geneesk. **77**:4917 (Oct. 28) 1933.

A case of traumatic rupture of the spleen is reported in a boy, aged 8 years, in which there was a hemorrhage in the splenic region. The nature and location of the pain speak in favor of the author's opinion (*Nederl. tijdschr. v. geneesk.* **77**:171 [Jan. 17] 1933) that the mechanism of traumatic rupture of the spleen clearly shows two stages.

VAN CREVELD, Amsterdam, The Netherlands.

## Miscellaneous

PENETRABILITY OF TRANSPARENT MATERIALS FOR ULTRA VIOLET RAYS. R. G. BLOCK and D. J. HOIJER, J. Lab. & Clin. Med. **18**:500 (Feb.) 1933.

The authors found that pure quartz glass and cellophane 0.03 mm. thick allowed the greatest amount of penetration of ultraviolet rays. Ordinary window glass and vitaglass were distinctly inferior to the former mediums.

McMURRAY, Galveston, Texas.

THE DISCARD OF THE CRADLE. JOHN ZAHORSKY, J. Pediat. **4**:660 (May) 1934.

The author disagrees with the present day practice of advising parents not to rock their babies or to pick them up when they cry. Playing with the infant after the evening feeding is not considered by the author to be such a harmful procedure as some persons teach. He believes that the great fear of overstimulation should be placed in the same category as that of overfeeding, which was expounded with emphasis only a decade ago. A new precept is proposed: "An infant during the first year of life should be amusing and should be amused." It is considered safe to leave the question of the amusement of children to the parents. A plea is also made for the old-fashioned cradle, as an aid in relieving annoying digestive symptoms. The author states: "In my opinion the total discard of the cradle has at least aided in two unfortunate results: first there has been a definite increase in bottle-fed babies and second an increase in umbilical hernias."

LEMASTER, Evanston, Ill.

A Criticism of Ante-Natal Work. A. J. Wrigley, Brit. M. J. **1**:891 (May 19) 1934.

A review and criticism of antenatal work are given, and the following conclusions are arrived at:

Those who practice the art of midwifery have seen that antenatal supervision as it is being carried out today cannot accomplish all, or even half, of what has been claimed for it. The pendulum has swung too far. The search for the abnormal has masked the preservation of the normal, and the pregnant woman receives a mixed blessing. There is probably more significance than was intended in the words of the final report of the Departmental Committee on Maternal Mortality: "It is essential that anté-natal care should reach the highest possible standard of excellence."

Royster, University, Va.

Metal Poisoning of Nurslings: Case Report. F. M. Messelevski, Arch. de méd. d. enf. **36**:678 (Nov.) 1933.

Among the conditions commonly cited as a source of nutritional disturbances in infancy, chronic metallic poisoning has received scant attention. Only two cases of poisoning by copper coins could be found recorded in the accessible literature, the first being an instance reported by Senfft in 1865 and another cited by Levine, without a date. A further observation is that reported by Messelevski before the Ukraine Institute of Bacterio-Epidemiology and Serology. An infant, 5 months of age, ingested a copper coin (a 2 copeck piece) which remained in the alimentary tube from Sept. 10, to Oct. 30, 1931, a period of fifty days. The prolonged action of minute doses of copper provoked characteristic symptoms of metal poisoning, such as profound disturbances of metabolism, retarded nutrition, albuminuria and vasomotor phenomena, all of these disappearing promptly on evacuation of the foreign body.

Amesse, Denver.

Abuse of Horse Serum Administered by Mouth. Georges Schreiber, Bull. Soc. de pédiat. de Paris **31**:70 (Feb. 21) 1933.

Two cases of sensitization to horse serum given by mouth are cited. The use of horse serum, orally administered, in selected cases in which excessive bleeding is considered probable, is subscribed to, but the indiscriminate administration of horse serum preoperatively, as practiced by some otolaryngologists, is condemned.

Benjamin, Montreal, Canada.

Transfusion of Blood in the Child. A. Muggia, Riforma med. **49**:1812, 1933.

Muggia discusses blood transfusion in children and makes a plea for its wider application. In the new-born, umbilical hemorrhage, melena, severe anemias and acute infections are indications for blood transfusion. For the older children the indications are the same as for the adult. In the endocrine disturbances of childhood the author believes that the repeated transfusion of small quantities of blood may be of value, since the donor's blood contains relatively large amounts of the endocrine hormones in normal proportions.

Rosi, Chicago.

Measurements of the Body Surface Compared with Indirect Estimations of the Same. B. Trambusti, Riv. di clin. pediat. **32**:53 (Jan.) 1934.

The author defends the direct measurement of the body surface as being more accurate than measurements calculated from various formulas. He uses an apparatus devised by himself for measuring the body surface.

Higgins, Boston.

TEACHING MOTHERS AND PERSONS AT LARGE IN THE BABIES-SAVING CAMPAIGN. G. A. ALFARO, Bol. Inst. internac. am. de protec. a la infancia **7**:179 (Oct.) 1933.

The author is persuaded that teaching the principles of health, to mothers in particular, is the most efficient means of fighting infant mortality. Through his research studies he has come to the conclusion that the ignorance of mothers in regard to the most elementary rules of child hygiene and child care stands foremost among the serious causes of infant morbidity and mortality and is considerably more important than poverty and dependency.

Hence the most efficient means of teaching mothers should be searched for, the social visitor being considered the center of such education, provided that she is conscious of her rôle and is socially inspired. Next comes the school; it might hold the first place, if school teaching were not so scientific and so unpractical; many older girls leave school without having profited by the teaching of child care.

Lectures and familiar talks in the dispensaries and clinics, aided by practical demonstrations, are valuable, if carried out with intelligence and common sense. Mothers should be taught not to injure their children by giving them improper or bad food. But nothing is comparable to the work that may be done by the influence of a sympathetic, tenacious social visitor; her teaching has the advantage of being practiced in the home itself and at the time when the mother feels the need of advice and is willing to follow it.

AUTHOR'S SUMMARY.

THE RÔLE OF THE FAMILY IN THE LIFE OF A CHILD. HANNA GRÜTERS-KÜHN, Gesundhfürs. f. d. Kindesalt. **8**:245 (Nov.) 1933.

This article, written in a charming style and betraying considerable personal experience, reviews the rôle which family life plays in the life of the child. The author returns to older conceptions when she points out that no institutional or group training can supplant the natural benefits which are afforded children by the father, mother and other members of the family. The result of attempts to train children in social incubators has ended in a complete reaction. The family remains the most important element in the child's life. Every attempt to supplant it, no matter how beneficently meant or how well organized, must be a mediocre surrogate.

REICHLE, Cleveland.

CURATIVE EFFECTS OF ATMOSPHERIC AND CLIMATIC INFLUENCES ON CHILDREN. LEOPOLD MOLL, Jahrb. f. Kinderh. **138**:179 (Feb.) 1933.

This article is a general discussion of the benefits of atmosphere and climate on the health of children.

CLAUSEN, Rochester, N. Y.

TEMPORARY DISHARMONY OF PHYSICAL DEVELOPMENT IN CHILDHOOD. IWAN ROSENSTEIN, Kinderärztl. Praxis **4**:18 (Jan.) 1933.

Rosenstein cites numerous cases (illustrated by photographs) of overnutrition, malnutrition and delayed skeletal and sexual development which after puberty return to normal without treatment. He thinks that the recognition of nature's power to regulate growth and development will guide physicians to make a better prognosis and, what is more important, prevent the institution of drastic forms of treatment.

HUENEKENS, Minneapolis.

INTRATHORACIC AND INTRA-ABDOMINAL IRRADIATION WITH THE QUARTZ LAMP. C. FERVERS, München. med. Wchnschr. **80**:1585 (Oct. 13) 1933.

Fervers describes a method of treating pulmonary and abdominal conditions by direct exposure to ultraviolet rays from the cold mercury vapor lamp. The technic is simple and requires no special surgical training. For the treatment of pulmonary lesions pneumothorax is necessary, and any fluid present is aspirated.

Under local anesthesia a trochar is inserted into an intercostal space, and the radiations from the lamp are passed through the trochar. For intra-abdominal irradiation ascitic fluid is removed, a pneumoperitoneum produced and the radiation is passed through in the same way as into the thoracic cavity. There have been no complications. The same patients have been treated twice within ten hours. So far the results have been encouraging.

BRAHDY, Mount Vernon, N. Y.

INFECTED TEETH AND THE CARE OF TEETH IN CHILDHOOD. HANS PICHLER, Wien. klin. Wchnschr. **46**:217 (June 30) 1933.

Pichler writes that the care and treatment of teeth during childhood is of great prophylactic significance, and that more can be done to remedy the diseases and defects of teeth during childhood than during later life. Because of the possibility of treating large shifts, dental care in the schools is the most important phase of dental and oral hygiene, and much can be accomplished by such treatment. However, treatment may be too late, since irreparable injuries frequently occur during preschool age. Since it is impossible to enforce seemingly obligatory care of children during preschool age at present, the pediatrician can contribute considerably to prophylaxis. Wherever possible, even prior to the appearance of manifest infections of the milk teeth, he should insist on regular dental examination and treatment.

ABT, Chicago.

NATURAL SELECTION DURING THE PERIOD OF EARLY INFANTILE DEATH. VON PFAUNDLER, Ztschr. f. Gesundhführung **1**:1 (Jan.) 1934.

The author studied the period of early deaths in order to discover whether these deaths should be avoided or are of value in eliminating the unfit. He calls attention to the extreme frequency of fetal death, which far surpasses the death rate of extra-uterine life. Deliberate abortion accounts for a large percentage of these deaths. Pfaundler, however, believes that this in itself is of social value in that the offspring of persons whose social character is often undesirable are eliminated. It is furthermore known that spontaneous abortion is frequently associated with congenital abnormalities. He sees in this process, therefore, a selective factor of high value for the race. Deaths at birth and shortly after birth are frequently due to disturbances of the mechanism of birth. However, even here the environmental factors do not entirely replace the congenital factors, since in this fashion again progeny unfit for birth are eliminated. Finally, he discusses the period of infancy. Here factors lying outside the germ plasm play a preponderant rôle. It is evident, however, to all pediatricians that under more or less equal conditions definite differences in resistance of different children show themselves. Hence, here, too, the death rate may be an expression of the elimination of the unfit. Finally, he calls attention to the fact that social service, far from interfering with this process, may aid it, since the indolent will not seek the aid of modern medicine or refuse its helping hand, whereas the intelligent will do so.

REICHLE, Cleveland.

DETERMINATION OF THE MATURITY OF YOUNG PERSONS. W. ZELLER, Ztschr. f. Gesundhführung **1**:13 (Jan.) 1934.

The author offers a series of criteria by which the sexual maturity of boys and girls can be determined. With the aid of these criteria he has studied the development of the various body dimensions.

REICHLE, Cleveland.

NORMAL THORACIC CIRCUMFERENCE OF THE SCHOOL CHILD. G. SARDEMANN, Ztschr. f. Gesundhführung **1**:118 (March and April) 1934.

This article presents the results of an extensive study on the dimensions of the chest. There are a number of tables.

REICHLE, Cleveland.

# Society Transactions

## PHILADELPHIA PEDIATRIC SOCIETY

*Meeting, Jan. 9, 1934*

JOHN DONNELLY, M.D., *President, in the Chair*

A CASE OF ACUTE DIVERTICULITIS. DR. PHILIP S. BARBA.

An infant had three severe hemorrhages from the bowel, the first at 8 months, the second at 9 months and the third at 11½ months of age. Each was preceded or accompanied by vomiting, with no blood in the vomitus. The last two were preceded by slight evidence of discomfort for from twenty-four to forty-eight hours.

Physical examination gave negative results on each occasion, except for the secondary anemia and shock immediately after the hemorrhage. A roentgenogram showed no abnormality. After the third hemorrhage the child showed signs of localization for the first time. These consisted of tenderness, decreased peristalsis and rigidity of the right rectus in the region of the umbilicus. Consultants agreed on a preoperative diagnosis of Meckel's diverticulum, with a perforating ulcer.

At operation this was found to be the condition. A mesenteric vessel at the site of the ulcer was the source of the preceding hemorrhages.

DISCUSSION

DR. J. H. MCKEE: The title of this paper suggests an exceedingly interesting episode. When the late Dr. J. William White went to the Mayo Clinic to be operated on he was convinced that he had carcinoma of the sigmoid flexure. Two days after he was operated on he received a letter from his friend, Sir Frederick Treves, in which he was told that he did not have carcinoma but that he had diverticulitis. I never think of the term without being reminded of that instance as Dr. White recorded it.

The study which Dr. Barba made of this patient enabled us to entertain at least a scientific suspicion before the patient was seen. Over the telephone we discussed the question of Meckel's diverticulum. Pathologically, we were aided by having two important specimens: gastric tissue, probably thus accounting for the diagnosis of ulcer and perforation, and also pancreatic tissue, which accounts for tumor formation in later life in some patients who show no symptoms. A number of physicians asked, "How did you make the diagnosis?" After carefully going over the points that Dr. Barba and I had covered they said, "Oh, it was a diagnosis by exclusion." Largely, it was, but not absolutely, because there were two prominent features, the symptoms in all patients with this condition who have symptoms—abdominal discomfort and hemorrhage.

I have seen this condition but four times. The first patient whom I saw, a girl, 17 months old, was seen with the late Dr. Robert P. Cummins. She had the remarkable history of having swallowed glass the preceding week. She had had two small intestinal hemorrhages. We wished to get her into the hospital. Dr. Cummins went over the types of glass that might be revealed by roentgen study. The parents delayed action, and one week later, when Dr. Cummins was called to see the child, she had a perforated ulcer and fatal peritonitis.

The second patient, a boy of 10 years, had been a big and sturdy boy until he showed symptoms of acute peritonitis. He was operated on by Dr. Babcock. The condition was found to be generalized peritonitis.

The third patient was seen in the Cooper Hospital in Camden, N. J. She was mentally deficient and had been suffering from abdominal pain for six weeks. Dr. Nassau did a wonderful operation. All the physicians were mistaken in the

diagnosis. The roentgenologist thought that the condition was perforated gastric ulcer. The stomach was enormously distended, but this was simply a part of the picture of intestinal obstruction. Dr. Nassau has this specimen at Jefferson Medical College Hospital. The girl lived five days after the operation, even though such a long time had elapsed before operation.

It is curious that the roentgen rays do not show anything in regard to this disorder. Dr. Pancoast said that he made a correct diagnosis of this condition once, but could not get any one to believe him. Most of the roentgenologists admit that their work is of no importance in the diagnosis.

There are a number of types of this disease. I wish that Dr. Wieder were present tonight to describe the condition from the surgical standpoint. He gave an interesting talk on the six types of Meckel's diverticulum when this case was reported at the Germantown Dispensary and Hospital.

DR. HYMAN I. GOLDSTEIN: This is an interesting report of a case. It was an unusual diagnosis to make correctly, particularly since the symptomatology during the course of events was atypical. The physicians were particularly fortunate in having made the correct diagnosis of diverticulitis of the ileocecal region. In the *New England Journal of Medicine* (**209**:1315 [Dec. 28] 1933) Homans and Hass of the Peter Bent Brigham Hospital, Boston, discussed "Regional Ileitis"—inflammatory, tumor-like thickenings of the intestinal wall, which have generally been given the name granuloma. Pathologically, the thickenings are decidedly suggestive of tuberculosis, particularly when they appear in the ileocecal region. There are ulceration and a penetrating inflammatory reaction involving all the coats. Much edema and fibrosis lead to thickening of the wall of the bowel and narrowing of the lumen. Perforation and fistula formation occur as complications.

Crohn, Ginzburg and Oppenheimer first separated regional ileitis as a definite clinical entity from the large nonspecific group of granulomas *Journal of the American Medical Association* (**99**:1323 [Nov. 16] 1932).

Clute, in *Surgical Clinics of North America* (**13**:561, 1933), reported several cases seen in the Lahey Clinic. The lesion, according to Crohn and his collaborators, should be situated in the lower part of the ileum just above the ileocecal valve, reaching exactly to that point and not beyond it. The condition is, perhaps, a nonspecific inflammation involving the mesentery and the wall of the last 12 or 18 inches (30.48 or 45.72 cm.) of the ileum.

This condition, regional ileitis, adenomatosis and diverticulitis must be considered in the differential diagnosis. Intestinal adenomatosis has recently been emphasized and must be considered in the study of these complicated conditions.

DR. J. P. C. GRIFFITH: This report takes me back to about twenty years ago, when I saw a case with some interesting features. The child had been earlier attended by a well known and able pediatrician, who made the mistaken diagnosis of pernicious anemia; I say this without any reproach, because I did not make the correct diagnosis either. I had no idea then of Meckel's diverticulum being possibly at fault, because at that time, so far as I recollect, the diagnosis of that condition had seldom, if ever, been made during life. In the early history, before I saw the child, there was no distinct report of hemorrhage, but only of extreme and increasing pallor. Some time after I saw the child there began to be intestinal hemorrhage and attacks of abdominal pain, but there was no localization or any sign of tumor or abscess; looking back, I cannot blame myself even now for not making a correct diagnosis. There was clearly ulceration somewhere, and hemorrhage was taking place, but with the little knowledge available at that time there was no reason to suspect Meckel's diverticulum. At autopsy there was found a perforated ulcer, no doubt a peptic ulcer, with walled-in inflammation.

I do not see that one has much chance to make a diagnosis of diverticulitis unless there is intestinal hemorrhage. A number of American authors have called attention to the significance of hemorrhage, but it does not occur in every case. In appendicitis there are decided tympanites, the well known site of resistance and signs of inflammation, while in diverticulitis the signs are probably nearer the umbilicus, and there is less likely to be tympanites. But, unless some of these

differential markings are present, I do not see on what one can early base a diagnosis. Later, matters may become clearer, as they did in the case reported this evening.

DR. PHILIP S. BARBA: When I reviewed the literature I found several cases described in which hemorrhage had been the principal symptom. After hearing a résumé of these cases one may wish to diagnose diverticulitis in any unexplained intestinal hemorrhage, but when one tries to persuade a surgeon to operate it is a difficult problem. All sorts of objections are made to opening an abdomen which gives no clues as to the site of the trouble, particularly in a child under 1 year of age and much weakened by severe hemorrhage. I should have liked to have an exploratory operation performed after the second hemorrhage, but the other physicians were unwilling. After the third hemorrhage the evidence was rather definite that the condition was Meckel's diverticulum and that an operation would be essential, but the perforation made operation necessary without waiting until we could build up the child's general condition.

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*Meeting, March 13, 1934*

JOHN DONNELLY, M.D., *President, in the Chair*

ANALYTIC REPORT OF THE EXAMINATION MADE BY THE CHESTER COUNTY HEALTH COMMITTEE. DR. R. T. DEVEREUX.

The findings in the examination of seven hundred and ninety-seven children from the largest families on financial relief in Chester County, Pa., supplied the data for this paper. The examinations were made by forty-five physicians. The results were unified and interpreted by chosen criteria, so that malnutrition was diagnosed by the nutrition picture and not by "average weight." Body build was considered in arriving at conclusions regarding weight. Almost 92 per cent of the children were of medium or slender build. With the use of body-build weight zones, 64 per cent showed the expected weight. Malnutrition appeared to be more of an individual than a familial problem. In 12 per cent of the families all the children were malnourished; in 58 per cent some were malnourished; in 30 per cent none was malnourished. Systemic disease correlated more closely with malnutrition than did underweight or disorders of behavior.

DISCUSSION

DR. H. C. CARPENTER: Dr. Devereux and the physicians in Chester County are to be congratulated. The correlation of malnutrition with expected weight and body build and with systemic diseases and disorders of behavior is most interesting. That only 1.5 per cent of all the children showed marked malnutrition is surprising. Pediatricians differ so widely in their appraisal of the individual child's nutrition that clinical examination has been of limited value for statistical purposes. A certain degree of unreliability in clinical diagnosis must be admitted. There is no satisfactory definition of malnutrition, and there is no standard index of malnutrition which is altogether acceptable. One must be exceedingly careful as to the interpretation of findings and figures relating to the incidence of malnutrition. I have gained the impression that the majority of interns in hospitals have a low standard for judging the state of a child's nutrition. A really well nourished child is seldom seen on the wards, and the longer one works in a hospital the lower the standard becomes. When an intern writes "Good nutrition" (as he usually does), I like to write after the words "Good for nothing." It would be interesting and of value if these children in Chester County could be reexamined with the arms-chest-hips index of measurement for comparative purposes. The Pennsylvania Emergency Child Health Committee of the Medical Society of the Commonwealth of Pennsylvania, under the leadership of Dr. Hamill, in gathering this information in regard to the health and nutritional status of Pennsylvania children in families

on relief, found 39 per cent with malnutrition in Fayette County. It is interesting to see that Chester County has about 30 per cent. This study should be concerned not only with children on relief, but also with those in the borderline families who are not on relief but are in great need. The problem varies in different localities, depending on the depth of the depression, the adequate character of facilities for relief and the medical care available. It seems regrettable that Philadelphia County was not included in this study. It would be a fine thing if the Philadelphia Pediatric Society offered its services to Dr. Hamill to carry out the investigation in Philadelphia.

DR. J. F. SINCLAIR: Dr. Devereux and the physicians of Chester County have done a fine piece of work. It is something that cannot be taken in all at once; a great deal of time is required to fully evaluate it. There is a great deal of detail, but it is well worth mastering. I hope that this report will be published.

DR. R. T. DEVEREUX: I might say that this interpretation was not made from the diagnoses of the individual physicians, but from the individual signs noted by them on the examination blanks. A great many physicians, even after observing the same signs, come to different conclusions. Only by using standard criteria for the signs which the physicians recorded were we able to make a unified interpretation.

Concerning Dr. Carpenter's remarks, 12 per cent of the children showed unequivocal signs of malnutrition, which was more or less severe, and 24 per cent showed slight signs—some so slight that it was controversial whether they were sufficient to indicate malnutrition. Every effort was made to include all the probable cases of malnutrition.

It is obvious that care must be exercised in comparing these figures with other published findings on malnutrition. Few of the published findings on malnutrition define the condition the incidence of which they claim to disclose, and often they prove to be figures for underaverage weight interpreted as being identical with malnutrition. It is well to remember, also, that moderate and marked malnutrition are the only nutritional states usually reported.

I agree with Dr. Stokes that it would be useful to compare the screening method used in determining the arms-chest-hips index for roughly detecting malnutrition with the Pennsylvania Emergency Health Examination. Personally, I think that the Pennsylvania standard is a reliable one.

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*Meeting, May 8, 1934*

JOHN DONNELLY, M.D., *President, in the Chair*

SOME ROENTGENOLOGIC AND ANATOMIC CHANGES IN THE GROWING SKELETON AFTER THE ADMINISTRATION OF BISMUTH AND ARSENIC. Presented by DR. JOHN CAFFEY.

A number of roentgenograms of long bones taken before, during and after the administration of bismuth and arsenic to children and infants in the treatment of congenital syphilis were presented. Bands of increased density in the growing skeleton, especially in the diaphyses, had developed during the therapeutic administration of these metals. The changes due to bismuth were more marked than those due to arsenic.

The shadows following bismuth therapy were seen to vary directly with the amount of bismuth given, and the duration of the treatment and also with the rapidity of the growth of bone. The level of the "bismuth line" or "bismuth zone" in the shaft was seen to depend on the time which had elapsed between the administration of bismuth and the taking of the roentgenogram. If the roentgenogram had been taken at or near the end of a therapeutic course of bismuth, the bismuth line was found at or near the end of the shaft. If, however, several weeks or months had elapsed between the bismuth therapy and the roentgenogram, the

bismuth line was found buried a variable distance in the shaft, with new and apparently normal bone between the bismuth line and the epiphysis.

Roentgenograms of long bones of three infants whose mothers had been treated during pregnancy (one with arsenic and two with bismuth) were presented. In each instance the results of both clinical and serologic examination of the infant were negative for syphilis and remained so. In the first case the mother had received bismuth intramuscularly during the last three months of gestation, and the bones of the new-born infant showed heavy bands of increased density at the ends of the long bones. In the second case the mother had received bismuth during the second, third, fourth, fifth and sixth months of gestation, and the bones of the new-born infant showed a heavy density in the middle three fifths of the shafts, which were ossified during the earlier months of fetal life while the mother was receiving bismuth. No changes were present in the bones of the new-born infant whose mother had received only arsenic therapy during the latter months of gestation.

Roentgenograms and histologic sections of young dogs that received massive doses of bismuth and arsenic over a three week period (from the sixty-eighth to the eighty-ninth day of life) were presented. Bands of increased density similar to those seen in infants and children had developed in the diaphyses of the experimental animals. The lesions due to bismuth were considerably more severe than those following the administration of arsenic. Histologic sections taken from the levels of the bones with the roentgen lesions showed a marked increase in the amount of trabecular cartilage and cancellous bone in these areas adjacent to the junctions of the cartilage and shaft. Roentgenograms and histologic sections of the lumbar vertebrae of dogs given bismuth showed a lesion similar to the lesions seen in the long bones, but less well developed.

Mention was made of the failure to produce lesions in the skeleton of newborn rabbits by the injection of bismuth and arsenic into their mothers during the last fifteen days of the pregnancy.

The similarity of the roentgenographic and histologic changes in the growing skeleton due to bismuth and lead was discussed.

DISCUSSION

Dr. Ralph S. Bromer: I believe that this is the first work of this character that has been shown. However, Dr. Caffey has added to the troubles of the roentgenologist, because there is now another dense line or widened zone of temporary calcification which must be differentiated from the same appearance caused by other conditions. The history and the medication of the patients whose bones are being observed in the roentgenograms must be known. Physicians have long recognized the increased density of the zone of temporary calcification in healing rickets and healing scurvy. Later we learned from Dr. Vogt, Dr. Park and Dr. Caffey of the increased density of this zone in lead poisoning, and now Dr. Caffey has added observations on the results of bismuth.

I cannot add anything to Dr. Caffey's paper, but I wish to emphasize again its importance. I know that I have overlooked this condition in films of patients undergoing antisyphilitic treatment. I should like to ask one or two questions. In the dogs and also in the patients who came to autopsy was bismuth recovered from the bones? Also, was bismuth excreted in the urine, and was it found in any other tissues of the body? Finally, I should like to ask Dr. Caffey's opinion regarding the mechanism of the deposition of the bismuth in the bones.

Dr. Henry H. Periman: One finds oneself in an embarrassing position when asked to discuss Dr. Caffey's paper, since there has been comparatively little in the way of research on the effects of the heavy metals on the growth of bone. The drugs belonging to the group of heavy metals resemble each other closely in their pharmacologic action. It therefore stands to reason that anything that bismuth does to the living organism is also accomplished by arsenic and antimony.

The prolonged use of arsenic in doses insufficient to bring about chronic poisoning was believed to have some effect on growth and nutrition, but actual research seemed to contradict this assumption.

Thus, Stockman and Greig found no change in the growth of animals which received arsenic over various periods of time. However, these investigators found that the only tissues affected were the growing bones, which appeared denser than usual. Sollman, feeding rats small quantities of arsenic, found that this drug tended to lessen the appetite and retard the growth of the animals.

The effect of arsenic on nutrition and longevity is both interesting and contradictory. The Styrian peasants are said to live to an old age, and believe that the continued use of arsenic enables them to live longer, work better and climb the mountains with less effort and less respiratory distress. On the other hand, the miners of Reichentlein, who are constantly exposed to arsenic, due to the fact that this element is contained in considerable quantities in the ore, are short-lived, very rachitic as children and in later life have ascites and respiratory diseases.

Bastedo stated: "In growing animals of poor nutrition arsenic tends to bring about an increase in the density of bone, the cancellous portion being encroached upon by the increasing thickness of hard bone." This, he believes, may be due to the increased vascularity of the bone marrow. In adults there is probably no effect on bone.

Dr. Caffey's contribution proves definitely that arsenic does have some effect on the growth of bone.

DR. MITCHELL RUBIN: Is there any definite order of deposition of this increased trabeculation?

DR. EDWARD THORPE: Have any other substances besides bismuth, phosphorus and lead been found to increase the density of the bone?

DR. JOHN CAFFEY: Because of the lack of time I have purposely omitted a review of the literature on both arsenic and bismuth. More than fifty years ago Gies demonstrated anatomically the increase in cancellous bone at the ends of the growing shafts after the ingestion of arsenic by experimental animals. Interestingly enough, he also found increases in the amount of fetal bone when a pregnant animal was fed arsenic. Recently Vogt found changes in the skeleton, which he thought could be attributed to bismuth given in the treatment of congenital syphilis. Last year Park and his co-workers, in a consideration of the differential diagnosis of lesions in the skeleton due to lead presented two cases with changes at the ends of the long bones in infants who had received bismuth.

It seems highly probable that the changes following the administration of arsenic and bismuth are similar to those due to lead and to a number of other etiologic factors. The changes are readily seen both histologically and roentgenologically, but—and I think that this is worth considerable emphasis—it is not possible for the roentgenologist or histologist to make an exact etiologic diagnosis from the roentgenologic or histologic appearance alone. The earlier a patient or an animal is examined the less well developed and less evident the lesion will be.

I know of no studies on the effect of antimony on the growing skeleton.

There are no data on the excretion of bismuth in this series of experimental animals.

A CASE OF RETICULOSIS. DR. BERYL H. PAIGE.

A case of reticulo-endotheliosis in a young child was reviewed, with a description of the clinical and pathologic findings, and nine cases of the disease in young children were summarized. Seven were cases reported in the literature and two were cases from the Babies Hospital.

The patients included four boys and five girls, whose ages ranged from 3 months to 2¾ years. The initial symptoms varied. Fever was present in eight cases. Purpura was found at some time in the disease in all nine patients, varying from punctate hemorrhages on the trunk to a profuse, purplish red, petechial eruption over the trunk, neck and extremities and petechiae in the gums, tongue and buccal mucosa. Splenomegaly was present in all cases and hepatomegaly in eight; lymphadenopathy was absent, slight or extreme and was localized or generalized. Roentgenograms demonstrated osseous changes in three instances. Eight patients had anemia

varying in type from mild to severe. The white cells ranged from 3,900 to 26,000 per cubic millimeter, with from 34 to 83 per cent polymorphonuclears and from 1 to 10 per cent monocytes; platelets numbered from 47,000 to 1,480,000. The duration of life after the onset of symptoms was from three weeks to one year. In only one case was the diagnosis made before death; this was made from biopsy of a small piece of spleen.

DISCUSSION

DR. RALPH M. TYSON: One wonders whether this is a new condition or an old friend in a new dress. Dr. Paige stressed the point as to whether this disease is the result of infection. All physicians are aware of the peculiar form that infection takes in the body. There is scarcely any satisfactory explanation for the form in which tuberculosis occurs in man. Why is it that the brain is involved in certain cases and the meninges, kidneys or bones in others? It is known that infection may have a rather lasting effect on the bone marrow and that often this effect may last long after the infection has cleared up. It is entirely possible, in my opinion, that the reticulo-endothelial system may be influenced by severe infection in a similar manner. Surely this case is one representing a number of severe infections. Its relation to other diseases of the reticulo-endothelial system and lipoid lipoidosis, as described by a number of investigators, is an interesting one, and it may be that this condition is a similar condition or combination of conditions. I am sure that most pediatricians who have seen a number of children die rather rapidly who had rashes similar to the one pictured by Dr. Paige will stop to think on other occasions and wonder whether they are dealing with a somewhat similar condition.

We have in Temple University Hospital a child brought there because of a foreign body, which had run through the soft palate into the nasopharynx. After the child had been in the hospital four or five days he showed an unusual temperature reaction and enlargement of the spleen and liver, and we were able to find streptococci in the blood stream; we have been under the impression that the case was one of infection of the blood stream. The enlargement of the liver and spleen suggested to me that perhaps we are dealing with an unusual condition of the reticulo-endothelial system. Dr. Paige has opened our eyes to a new field, and we are indebted to her for her splendid presentation.

DR. THEODORE S. WILDER: I have been wondering how many cases of this condition have been seen and mistaken for cases of thrombocytopenic purpura or some other condition. It is interesting that in these purpuric conditions the number of platelets remains high in some cases. I wonder whether the great varieties of infection in this condition are due to the presence of unusually virulent organisms or to the complete breakdown by apparently nonvirulent organisms. It would seem that the body response to infective agents suffers a complete breakdown much as one sees in the leukemias. As a matter of fact, this case is not unlike a case I recently saw which was diagnosed as monocytic leukemia.

DR. BERYL H. PAIGE: In regard to the statement that Dr. Wilder made of the similarity of this case to a case of monocytic leukemia, this disease has been regarded by some as an aleukemic phase of monocytic leukemia.

STABILIZATION OF THE DIABETIC CHILD. DR. JOHN M. BRUSH.

The methods advocated and the time required to stabilize the juvenile diabetic patient are extremely variable. Some clinicians believe that three months or longer are necessary and that then the instability of the diabetic child tends to defeat the end accomplished even in this length of time. Others allow the period of improvement to be spent in the home, thereby superimposing on what is at best a difficult regimen when first undertaken adjustment to insulin and the fear as well as the actual incidence of hypoglycemic shock. It is disturbing to a family to have the child have a convulsion on the way home from the hospital.

Attention will be directed toward the relatively untreated diabetic child, because in these cases only are the results of the method constant. It is my belief that the

parents of the child who has never been stabilized can be assured that within a period of four weeks or less of hospital treatment, irrespective of the apparent severity of the disease, their child will be discharged on a diet adequate for growth, approximating the normal in the distribution of food and sugar-free, on one dose of insulin a day of less than 10 and usually less than 5 units.

The principles involved in the method are as follows:

(First, I may state that none of the statements should be looked on as being applicable to the adult.)

1. Hyperglycemia, as reflected by glycosuria, is relatively harmful. Persistent marked improvement cannot take place during a period of constant spill.

2. Chronic hypoglycemia allows maximum improvement. This is unfortunately but unavoidably accompanied by frequent clinical manifestations of shock.

3. Increasing the diet as tolerance improves is detrimental to pancreatic recovery. This does not carry with it the implication that diabetes mellitus is pancreatic in origin.

4. Allowing the patient to desugarize over a period of days on a constant diet, with constant doses of insulin, is relatively harmful.

5. Too rapid reduction in doses of insulin interrupts improvement.

6. An excess of calories markedly increases the insulin.

The method is extremely simple:

1. As soon as recovery from ketosis has taken place the full maintenance diet is offered and is usually taken within forty-eight hours after admission.

2. The amount of insulin is rapidly raised to the desugarizing level. The usual three doses are given, and in addition an injection at 4 a. m. is also used. An average distribution would be 40, 10, 20 and 4 units.

3. As tolerance improves, as manifested by shock, the amount of insulin is decreased as slowly as is compatible with safety.

There are several advantages in this procedure: It sharply limits the stay in the hospital; the goal is clearly defined, and there is little of "feeling one's way along." The one important consideration is that the child is delivered to the home in a stable and mild diabetic state, sugar-free, on such a small dose of insulin that shock is not to be feared.

Diabetes mellitus in childhood, however, is a chronic disease in which recovery is practically unknown. Treatment should be directed toward producing the best results at the end of five or twenty years. No evidence is available that the regimen described is of any value in modifying the ultimate outlook.

DISCUSSION

DR. JOHN P. SCOTT: I had two or three years of practice in the pre-insulin period in the history of diabetes. I remember what a hopeless and discouraging disease it was. Later came a period in which insulin was not used unless such treatment was unavoidable. With this the allowance of fat was run up to a high level. I remember well a child of 16 months, untrained and incontinent, whose diet, high in fat and low in carbohydrate, was managed with great difficulty in order that a small amount of insulin might suffice. The liberal use of insulin and carbohydrate has greatly facilitated getting children stabilized, but one still has trouble with irregularities and wide swings in the blood sugar content. For example, a child may have a sugar-free urine for twenty-four hours and a blood sugar content of 250 mg. per hundred cubic centimeters at 7 a. m. and go into shock at 11 a. m. Or the child who is sugar-free on a diet for three or four days may begin to show sugar without a good reason. Several factors besides infection account for these irregularities. One is activity. The child who is in bed does not have a fixed caloric expenditure; on some days he is quiet, and on other days he may be in constant movement about his bed, jumping, walking, doing acrobatics or fighting with a child several feet away. A great deal depends on the stimulating propensities of the other children and on the influence of the nurse or nurses who take care of the children. Emotional influences may be present and alter the

glycogenolytic function independently of activity. Among these are visits from parents, examinations, treatments and seeing other children go home first. It seems to me that one sees more shocks than previously. Dr. Brush has brought to our attention how casually one can take these shocks. Physicians have always been somewhat apprehensive about shocks and have been anxious to avoid them; we do not want to put our hospital even in the position of the man who had to stop a trolley-car to get a lump of sugar. Much less do we want to have the alarming occurrence of shocks in the home, for often children are taken on picnics and excursions where it may be difficult to attend to such matters. In spite of this apprehension, Dr. Brush is right in saying that these shocks can largely be discounted. Judging from the method of Dr. Brush and its prompt production of a low level of blood sugar, the maintenance near a shock level certainly seems to hurry up the patient's eventual stabilization and discharge.

Dr. D. Stewart Polk: Dr. Brush's presentation, describing this method of cutting down the amount of insulin required to an unusually low figure, is a fascinating one. In Philadelphia we usually employ a much more conservative method, giving a diet at first somewhere near the basal metabolic requirement and increasing it about 10 per cent daily during the first three or four days as long as the urine remains free from sugar and the blood sugar level remains fairly low. I do not believe that our children leave the hospital as soon as those under Dr. Brush's treatment do. On the other hand, patients with mild cases are dismissed within less than twenty-one days. I think that all agree on approximately the same number of calories for the child. We probably agree on the protein and carbohydrate level. Almost every physician is giving more carbohyrate now than formerly; the average amount is somewhere around 135 Gm., with as much as from 165 to 180 Gm. for the late years of childhood. I am particularly interested in following these patients in the dispensary, and I wonder how long they can get along on a single dose of insulin. I have to give two or three doses to keep them sugar-free through the day. One mother decided that her child was getting a little too much insulin and omitted insulin for three days. The first day after the omission the child showed no sugar. The second day she showed a precipitate, and the third day all the specimens showed a great deal of sugar. The mother quickly put the child back on insulin given three times a day. Our children are best controlled by insulin given three times a day. We find that a weighed diet is far superior to a measured approximation.

I should like to try Dr. Brush's method, as it sounds interesting. It seems to save time, and yet the occurrence of shocks so frequently is disturbing, especially in children 3 years of age or less whose veins are not easy to enter.

Dr. Rustin McIntosh: I think that I should confess the skepticism with which I greeted Dr. Brush's procedure when I first became acquainted with it in the fall of 1931. But after watching the results I have become thoroughly convinced that it is worthy of continued use. I should like to point out again the policy on which his method of treatment depends: One tries to give the patient as much insulin as one safely can, rather than the smallest amount on which he might get along. It is as though one made an effort to put the pancreas at rest over a period of several days. In the end convalescence is hastened; the patient goes home requiring much less insulin, and the whole plan of treatment is simpler. Formerly, patients went home and were given 20, 30 or 40 units; their tolerance then improved, and they would get insulin shocks, with which they were not familiar, under conditions that made them not easy to deal with. The patients now leave much better stabilized.

Dr. John M. Brush: The question was asked as to how long a patient should be given a single dose. Sometimes I discontinue it entirely. As a rule, I expect a patient to continue on a single dose for a year and then decrease the dose, with a drop in tolerance; during the third year I expect the condition to remain constant. Some patients do worse and some better. With regard to shock on the wards, as a rule it is not alarming; the nurses become extremely clever in noticing anything wrong with the child and give him orange juice. We have not had to resort to

intravenous injections of dextrose in more than fifty or sixty cases of shock, at the most, not any more than three or four times a year. There is a difference between shock in the hospital and in the home. When the patient gets home all attempts are made to prevent him from having shock, so I reduce the amount of insulin promptly.

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*Meeting, June 12, 1934*

JOHN D. DONNELLY, M.D., *President, in the Chair*

BIOLOGIC EFFECTS OF THYMUS EXTRACT. Presented by DR. L. G. ROWNTREE, DR. J. H. CLARK and DR. A. M. HANSON.

White rats (Wistar strain) were given intraperitoneal injections of an extract of thymus, prepared by Hanson, daily for nine months. In the first generation ($F_0$) treated the test animals were heavier, bred more frequently and had larger litters of heavier average weight per rat. The control animals cast five litters, totaling twenty-two rats; the thymus-treated pair, nine litters, totaling eighty-six rats. Little of note appeared in the second generation ($F_1$), except somewhat more rapid development, somewhat earlier eruption of teeth and earlier descent of the testes (within from twenty-two to twenty-nine days). This tendency became more pronounced with succeeding litters. In a seventh litter the animals were larger; the body hair was visible almost from the beginning, and teeth erupted on the second day.

The third generation of thymus-treated animals ($F_2$), as judged from forty-three surviving rats, evidenced rapidity of development almost beyond belief. The average weight at birth was 6.5 Gm., while that of the controls was 4.3 Gm. (eighteen animals). Teeth appeared and the ears opened on the first and second day. The animals were completely covered with hair and the eyes opened on the fourth and sixth day. The testes descended on the twelfth day, and the vagina opened on the twenty-third to the thirty-first day. Estrus was established from the thirtieth to the fortieth day. The female of one pair ($L_2F_1$), which had been given injections from birth, cast a litter at forty-two days. The curves for growth of the third generation of thymus-treated rats, compared with those of controls, revealed marked acceleration, with the maximum difference about the sixteenth day, and approached each other about the sixtieth day. The treated animals appeared unusually docile and contented.

The effects of treatment (acceleration in the rate of development) transmitted through the grandparents and parents is much more marked on the offspring (third generation) than those resulting from continuous treatment for the same length of time confined to parents alone (the seventh litter of the first generation).

It is evident, therefore, that this thymus extract has accelerated growth, development, maturity and fertility. The effects of thymus extract become progressively more pronounced as treatment is continued into succeeding generations.

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## CHICAGO PEDIATRIC SOCIETY

*Regular Meeting, Oct. 16, 1934*

STANLEY GIBSON, M.D., *President, in the Chair*

PHYSIOLOGIC CHANGES OF ADOLESCENCE. DR. ARTHUR H. PARMELEE.

Puberty is a period of life between childhood and adult age characterized by certain unique anatomic and physiologic peculiarities associated with and accompanying the process of maturation. The differences in the physiology and pathology of this period are sufficient to justify the recognition of it as deserving

special study. At present there is a surprising paucity of dependable information about puberty. Practically, the period of adolescence is a "no man's land," so far as the medical specialties are concerned. The main distinguishing feature of pediatrics as a specialty is that it has to do with the human organism in health and disease under the influence of the phenomena of growth. It would seem, then, that the background and the traditions of pediatrics, of all the specialties, should best fit in with the problems of puberty, a period in which rapid growth and development are the outstanding features.

The real source of the puberty impulse is probably the anterior lobe of the hypophysis. This gland also acts as a governing center in the complicated correlation of hormones from the various endocrine glands. While the endocrine glands are of the greatest significance because of their stimulating and intensifying influence, they do not primarily and inevitably determine growth, but act rather as auxiliary organs, helping to carry through the life rhythm stipulated by the heritage of the individual.

The growth impulse of puberty as it affects the separate organs parallels to a certain degree that of the entire body.

The metabolism shows an increase appropriate to the increased growth impulse, the increase of body mass and its organization.

The pathology of puberty includes the various deviations from the natural course in the orderly progress of development.

Statistics on mortality show that the lowest figures occur for the age of the onset of puberty. The mortality curve for the infectious diseases reaches its low point at the age of maturity, but for tuberculosis this curve rises sharply during puberty.

The chief objects of this paper are, first, to call attention to the fact that the physiology and the pathology of puberty are distinctive mainly because of the influences of the phenomena of growth peculiar to that age and, second, to suggest that the problems of puberty rightfully should be a pediatric responsibility.

PSYCHOLOGIC PROBLEMS OF THE ADOLESCENT. DR. BERT I. BEVERLY.

Adolescence begins at puberty and is the period of adjustment of the individual to society. The ability to do abstract reasoning is essential to this adjustment. The chief changes in the mind of the child at this age are marked exaggeration of sexual impulses and sexual attraction. The attempt to adjust the instinctive emotional impulses from within to the norms of society brings up many problems in the minds of all individuals. Those problems are: (*a*) responsibility for sexual behavior (influenced by early training in sex hygiene); (*b*) the gaining of independence (influenced by early methods of discipline); (*c*) weaning from the parents (influenced by early spoiling); (*d*) sensitiveness and feelings of inferiority (influenced by early scaring about being bad or dirty); (*e*) individual fears, varying with the person, and (*f*) environmental and physical difficulties, varying with the person.

Inability to make adjustment brings about behavior problems, such as outbreak of temper, failure in school, truancy and various forms of so-called nervousness. Treatment is, first of all, prophylactic, which indicates radical changes in the rearing of children. Persons unable to make an adjustment should be considered mentally sick and in need of medical psychiatric care, which can best be carried out if the patient is removed from the home.

DISCUSSION

DR. C. A. ALDRICH: In these papers, which so admirably sum up the problems of adolescence, so much has been said that I am dismayed at the idea of competent discussion. Not only am I dismayed at this prospect, but consideration of the topic during the past two weeks since I have known that it was my lot to appear on this program has engendered a certain humility of spirit which makes me hesitate to preach in any way about adolescence.

When I look about at my own generation and its follies culminating in the World War and its massacre of adolescents, and when I reflect on the subsequent nineteen-twenties in which many of the lasting human values in life were brushed aside in a mad chase for temporary acquisition, I feel more like asking youth to show us a new way than instructing it in its own way of life.

In his book "Adolescence" Frankwood E. Williams states that adult accomplishment is characterized by the fact that the individual ceases to be ruled by his emotions and is controlled by reason. Measured by this yardstick, we must admit that our generation is still almost entirely adolescent. As illustrations, witness our hysterical war emotions, the astounding effect of meaningless political slogans, the emotions aroused in parents by the natural acts of children and our own immature sex reactions. The task, then, of carrying the present crop of babies through childhood into normal adolescence and into complete adult accomplishment is immensely complicated by the fact that the present adult population has not yet grown up to the point at which immature emotions are controlled by reason. The blind is leading the blind.

I would not paint this gloomy picture of the situation if there were not, in my opinion, something definite for pediatricians to do about it. Perhaps, more than any other group of physicians in the world, we have obtained the public confidence, due to outstanding successes in nutrition. Hardly a newspaper or magazine is printed which does not emphasize the large proportion of persons who accept our word as law. Furthermore, we have the immense strategic advantage of being able to meet the problem of child nurture at its inception, during the newborn period. It seems to me that if we could adequately show parents that their greatest opportunity lies in providing the proper environment for their children's growth rather than in dictating their behavior, and if we could agree on sensible objectives for children's development, we might be able to fertilize the soil so that healthy adolescence might emerge later on.

H. C. Miller, in his book "The New Psychology and the Teacher," gives the following summary of the ideal development of the child: "He begins life entirely dependent, egocentric, irresponsible; he should become fully independent, altruistic, responsible. He has to pass from the completely filial to this completely parental attitude. From being the victim of circumstances and environment, helpless in the face of these two factors, he should end by being independent of both and the captain of his own soul. Lastly, from being unconscious and then more and more conscious of himself as a center of attraction, he should attain the complete adult attitude which includes the readiness to be ignored." Such attainment is necessarily a product of growth and cannot result from repression.

Frankwood E. Williams, in "Adolescence," discusses the adult attitude toward growth as follows: "Parents who are no longer slaves themselves will have no need to place others in bondage. Their satisfaction will be not in moulding but in watching the unfolding and development of the possibilities of the child itself. They will know and will furnish to the child such emotional elements as the child needs for its nourishment, and for which the child is dependent upon them—neither surfeiting him nor starving him, nor doing first the one and then the other. They will watch with interest and without alarm the experiments of the child as it endeavors to discover itself and its world, satisfied themselves to serve as mentors. They will watch with equanimity, as long as it is healthy, the development of a personality and character peculiarly the child's own, even though it may be one they would not personally have prescribed. That it is healthy and sound, and that it is the child's own, will be sufficient, a character that the child can have confidence in and respect and which they can respect, and respecting, respect themselves as well.

"For they have done a good thing. By their union they have given creation to a new individual containing a grouping of potentialities that has never before precisely existed. They have protected it, nourished it, guided its energies without bruising it, into healthful channels and permitted it to develop its own peculiar potentialities to their fullest extent. They have created a companion worthy of themselves and of all men and women free to respect themselves."

This boils down to the fact that in the proper development of children healthful parental attitudes are all-important. These parental attitudes will develop largely in response to pediatricians' own attitudes toward growth. We must be thoughtful, tolerant of the child and almost reverent in regard to the age-old forces of growth working out his destiny. These are so much wiser than our wisecracks. I blush to think of some of the thoughtless statements that I have in the past made to mothers.

Adoption of this therapeutic attitude necessarily entails scrapping much of our routine technic. Babies will have to be treated individually rather than collectively, and emphasis must be placed on the specific points of excellence in each child rather than on his deficiencies, except, of course, in cases in which these require active treatment. Children should not be "picked on." Good points should be emphasized and defects brought out incidentally.

Pediatricians could, if they would, do more to promote healthy adolescence than any other group of workers. They would function, in my opinion, not so forcefully by their advice to young persons during the actual period of adolescence as in creating in parents during the infancy of their children adult attitudes toward growth. This would give adolescents a fighting chance to unfold more charming personalities and completely adult minds. Our problem is one of adult education, both lay and professional. The schools are taking the lead in this kind of education. The North Shore Country Day School, for instance, has a course in the primary grades in which sexual problems are treated frankly, and in the senior classes labor is explained to the girls.

DR. GEORGE J. MOHR: It is apparent that the problems of adolescence are comprehensive. My remarks, which are primarily derived from some formulations by Anna Freud, are intended to simplify some confusing features of adolescence. Adolescence is not the first period of rapid growth and active change that the child experiences. At about the age of 5 or 6 years the child is completing a period of exceptional growth and development, both physical and psychologic. He has just made a series of readjustments that carry him from an infantile state to that of the young school child, and he leaves behind him certain anxieties and uncertainties characteristic of early childhood. Following this comes a relatively latent period without abrupt or disturbing psychologic change. In adolescence the period of rapid growth and change is repeated in a manner that is somewhat determined by the nature of the earlier period of rapid growth, and the adolescent faces the inner difficulties that Dr. Beverly has stressed. Dr. Beverly stated that the child tends to be somewhat overwhelmed by the changing character of his sexual impulses, his social problems and his aspirations.

If one considers the characteristics of adolescence, it is safe to say that some of these are determined by the defenses presented by the adolescent in meeting his problems. For instance, every one is acquainted with the adolescent who is shy, shuns society and is rather unapproachable. His method of meeting his problems of adolescence is to attempt to put them off, to ignore and deny them. Since social relationships are difficult for him, he denies their necessity, and one recognizes the reserved, difficult and unapproachable adolescent. The same feeling on the part of the adolescent may, however, assume a different form of outward expression. A great egotism may mask the situation, the formula behind which is: "I cannot get along with my fellows; I will turn to myself entirely and need no one."

Dr. Beverly mentioned another characteristic of adolescence — idealistic tendencies, which may evidence themselves in any field of interest. The idealistic adolescent finds all the common forms of living adhered to by adults intolerable; he is critical and oversensitive and regards the practical adult world as sordid or unacceptable. In reality, he is none too certain about his ability to cope with adult sexual and social responsibilities, and his idealism is in part a denial of the desirability or necessity of so doing. A further tendency of the adolescent is to intellectualize; the stubbornly argumentative adolescent is familiar to every one. Here one notes an attempt to master anxiety about the problems that have been

mentioned through the fiction of an intellectual mastery of a situation in which the discomforts are really in the emotional field.

I believe that if one bears in mind that adolescent children feel put to it by the problems that they are attempting to meet, and that they indulge in defensive tactics, as I have indicated, one may have a clearer understanding of the characteristics of adolescence which too often are regarded by the parent, and sometimes by the physician, as the disagreeable traits of children at a "difficult" age.

DR. M. L. BLATT: Having listened to these interesting papers, one wonders how frequently adolescents are able to handle their own problems. With the help of fairly well-balanced parents, it is my opinion that many adolescents pass through this period with little difficulty. The subject is one of intense interest, because when such youngsters have difficulty in adjusting themselves to their social environment the solution of the problem requires great patience and much ingenuity. It is imperative that pediatricians recognize the seriousness of these problems so that parents may be properly advised.

DR. HENRY E. IRISH: Many of the problems that Dr. Parmelee mentioned are somewhat new to pediatricians. Unlike some pediatricians, I have been in the habit of seeing children at a somewhat later age than a pediatrician ordinarily would, principally because I have believed during a period of years that the friendship formed and the intimate knowledge of the physical history are too valuable to discard.

In relation to sexual development in girls, I think that they have been better analyzed and better handled by gynecologists who have made a study of this field and of the particular hormones indicated, and it has seemed to me wise to refer such patients to some of my friends who are gynecologists, who are better versed in these problems as the result of applying these substances to women. Perhaps with the new order it will be necessary for pediatricians to familiarize themselves with borderline problems.

A few weeks ago a 14 year old girl was brought to me with profuse menstruation. She had a rather low hemoglobin count, and the question arose in my mind whether I was fitted to take care of her properly. Pediatricians will probably practice general medicine among their patients as they feel competent to do, but will probably refer special problems relating to certain organs and systems to a specialist in that organ or system.

I like particularly one statement that Dr. Aldrich made in discussing the attitude of adults toward children; that was the suggestion not to "pick on" them but rather to pick out the things that they do well and to praise them highly for these things. I have found in my experience in raising five children of my own that the best weapon I have is excessive praise. It is surprising what effects can be secured by magnifying the things that they do well. I think that this often serves as a substitute, perhaps, for some of the things that are not quite so happy in their lives. I feel assured that if parents were taught to substitute praise and commendation for blame and condemnation much would be accomplished.

DR. CHARLES SCHOTT: The points that Dr. Parmelee brought out regarding the endocrine glands are interesting. Many of the members probably have an idea of some of them, but the way in which he described them made them very clear to me. The change in adolescence is normal, as Dr. Blatt brought out.

I was a little disappointed in the scope of the papers, particularly in that of Dr. Beverly's. He told the harrowing things, but did not give the approach to them. When should one begin to tell growing children that they are approaching a critical time or a changing time—that they are going to be little men and women instead of little boys and girls? Usually pediatricians tell them when the mother asks them to or when they believe that the child will grasp the situation. Recently I had two interesting experiences. A mother who lives on the North Shore, who attends "highbrow" lectures on psychology, brought her 11 year old boy to me and wished me to talk to him. He was just a little boy, and I had some difficulty in explaining things and finally said, "When you get a little older an urge will come over you—a sex urge, it is called." He sat listening to me, and I wondered

what impression was being made. I talked to him for a while and then sent him out. When his mother came in laughing a few minutes later I asked her what the joke was. She said that when the boy came out into the reception room he pointed to my secretary and said, "She is old enough to have the urge."

Another child was brought in who was further along in adolescence physically than the first boy, but who showed no psychic changes. I talked to him, but he did not know what I meant; it was like talking to a blank wall. Yet he showed the physical changes that go with adolescence.

Should the father, the mother or the physician make the decision as to when a boy and girl should be told about sex? I think that the physician should do it, for I believe that he can better judge what the child is able to grasp and understand than the parents, who often overrate or underrate the child's ability to understand things.

Adolescence is a normal thing. Most of us have conquered it, I hope, but some are probably still in the adolescent stage.

DR. JOHN A. BIGLER: I should like to ask Dr. Parmelee whether in retarded development, especially in girls with rapid physical growth, it does any good to give any of the glandular extracts, especially ovarian extract, with the view of bringing on menstruation.

DR. ARTHUR H. PARMELEE: I shall digress from my own subject to speak of some of the things that belong rightfully in Dr. Beverly's discussion.

In replying to Dr. Blatt's remarks, certainly there are many perfectly normal adolescent children. But there are also many children who have many problems and have to make a great many adjustments, but who are capable of making them if the parents give them an opportunity to do so. My opinion about the attitude of the parent toward the adolescent child is that it should be that of a silent but interested bystander waiting to see what will result from his previous handiwork. By the time the child has reached adolescence the parent has done practically all that he can, and most of the trouble with adolescent children, unless they have got into some terrible muddle, is either the fault of the parents, the schools or some one in their environment who has muddled the thing. I think that meddlesome interference on the part of the parent after the child is adolescent and wants to be independent does an enormous amount of harm. Parents should give advice when they are asked for it, but otherwise they should let the child work things out for himself. If they have done a good job up to that time by their own example and their sanity in handling the child, the child will be normal and will be able to make his own adjustments.

The remarks of Dr. Irish in regard to praising fine, superior qualities in a child and emphasizing those qualities were very apropos. That practice is not new or startling, but is the basis of all modern and progressive teaching. However, I disagree with Dr. Irish on the point that one should send children to the physician who is competent to look after the disturbances of puberty. As I said in my paper, as I looked through the textbooks of medicine, even the most recent textbooks on gynecology, aside from experiments that had been done and the conclusions regarding the effects of various hormones, there is little constructive help in regard to puberty. There is great difficulty in knowing to whom to send adolescent children. This field is a "no man's land," and I think that pediatricians should be the ones who should take care of children during adolescence, for during adolescence the things that are going on in the human organism are unique because of the influence of growth. We should and must be the best fitted to take care of these persons; it is a new field and an open field for us. Unless we take it over, it will be taken over by some one far less fitted than we are.

In regard to Dr. Bigler's question concerning delayed adolescence, I think that in many cases glandular therapy is indicated, but many cases in which adolescence is somewhat retarded are still within the limits of normal, and to try to hurry up adolescence does no particular good to the child.

Dr. Schott spoke of one child who thoroughly understood what he was told and of another who did not know what he was told, although he was physically

better developed. This indicates, I think, that the condition of a child's consciousness of sex, his information regarding sex or his mental sexual development is entirely a matter of the totality of his own sexual experience up to that time. One cannot give him sexual experience by talking to him about the sex organs.

DR. JOHN A. BIGLER: I wish to know whether one can bring on menstruation in adolescent girls by giving ovarian extract.

DR. ARTHUR H. PARMELEE: I do not know, but I think that experimental work with extract of the anterior lobe of the pituitary from the urine of pregnant women has shown that menstruation can be stimulated. Whether it is wise to do this I cannot say.

DR. BERT I. BEVERLY: It does not seem to me that one gets very far in trying to decide what is normal. I tried to emphasize that all adolescents have problems varying in severity and complexity, and further, that the severity and complexity of their problems are determined in a large measure by early training and determine to a great extent their ability to adjust themselves.

In answer to the question as to when information regarding sex should be given, it seems to me that it should begin with an attempt to give parents the right attitude when their child is an infant and should be continued in much the same way as physical care.

Dr. Schott's two cases illustrate one difficulty in attempting to give information regarding sex hygiene to adolescents and emphasize the necessity for first gaining the confidence of the child.

# Book Reviews

**An Atlas of Infant Behavior: A Systematic Delineation of the Forms and Early Growth of Human Behavior Patterns.** By Arnold Gesell, Ph.D., M.D., Sc.D., Director of the Clinic of Child Development and Professor of Child Hygiene in Yale University. Volume 1: Normative Series, In collaboration with Helen Thompson, Ph.D., and Catherine Strunk Amatruda, M.D. Volume 2: Naturalistic Series. In collaboration with Alice Virginia Keliher, Ph.D., Frances Lillian Ilg, M.D., and Jessie Jervis Carlson, Ph.D. Leather. Price, $25 per set of 2 volumes. Pp. 921, with 3,200 illustrations. New Haven: Yale University Press, 1934.

These two books are magnificently prepared with red leather bindings and are in loose-leaf form, so that the material can be taken out and arranged in whatever manner one wishes to meet the peculiar demands of the situation. The introduction to the book gives a good description of its contents:

"This Atlas delineates the early growth of human behavior. The word atlas is used advisedly for we have attempted to chart in a systematic manner the major areas of the infant's behavior world. In these two volumes, embracing over three thousand photographs will be found visible manifestations of his maturing patterns of action and reaction: his bodily postures—supine, prone and ultimately upright; his space defying projections of posture—rolling, pivoting, squatting, crawling, creeping, cruising, climbing, walking; his sensori-motor incorporation of the physical environment through eyes, hands and mouth, staring, scanning, approaching, contacting, grasping, manipulating; his adaptive appropriation of this environment through self initiated and induced exploitations; his cumulative assimilation of the social forces which pervade this selfsame environment, his awareness of persons, his smiling, laughing, crying; his responsiveness to gesture, to speech, and to unremitting domestic ministrations; his compliances, resistances and participations in feeding, bathing, toilet, dressing, undressing, cleansing and physical transportations; his own spontaneous explorations and protean play activities; and finally his sleep—a paradoxical form of behavior which immobilizes him and yet propels him to a higher maturity level of performance on the morrow. Thus through the continuing strange eventfulness of a cycle of days the infant pursues a cycle of mental growth."

Twenty-four different behavior situations were included in a normative survey. All these are pictured in volume 1 serial groups of from four to eight pictures, each picture having a descriptive title, in the following order:

*A.* Posture and Locomotion
(Normative Crib Situations)

1. Supine behavior (4 weeks—40 weeks)
2. Prone behavior (4 weeks—56 weeks)
3. Stair-climbing (40 weeks—56 weeks)
4. Sitting (4 weeks—52 weeks)
5. Standing and walking (4 weeks—60 weeks)

*B.* Early Perception and Prehension
(Supine Situations)

6. Dangling ring (4 weeks—28 weeks)
7. Rattle (4 weeks—28 weeks)

*C.* Perceptual, Prehensory and Adaptive Behavior
(Sitting Situations)

8. Consecutive cubes (21 weeks—56 weeks)
9. Massed cubes (16 weeks—60 weeks)
10. Tower building (44 weeks—60 weeks)
11. Cup (12 weeks—36 weeks)

12. Spoon (16 weeks—36 weeks)
13. Cup and spoon (32 weeks—56 weeks)
14. Cup and cubes (32 weeks—56 weeks)
15. Pellet (12 weeks—56 weeks)
16. Pellet and bottle (32 weeks—56 weeks)
17. Bell (16 weeks—56 weeks)
18. Ring and string (28 weeks—56 weeks)
19. Ring, string and bell (32 weeks—56 weeks)
20. Paper and crayon (36 weeks—56 weeks)
21. Performance box (40 weeks—56 weeks)
22. Form board (20 weeks—56 weeks)
23. Ball play (40 weeks—56 weeks)
24. Mirror (40 weeks—56 weeks)

In all, there are more than three thousand illustrations in the two volumes. In the first volume, or the "Normative Series," Helen Thompson and Catherine Strunk Amatruda collaborated with Dr. Gesell. The illustrations in this volume may well be used as a standard for work along the line of infant behavior. They may be used for studying the various advancements along different lines. The vast amount of work which has preceded these volumes makes them authoritative in this particular field.

Volume 2, in which Alice Keliher, Frances Lillian Ilg, and Jessie Carlson collaborated with Dr. Gesell, emphasizes individual differences and is devoted to the naturalistic attitudes of six major cases. This volume contains about eighteen hundred photographs and depicts in detail various phases of the usual life of the infant, including breast feeding, spoon feeding, cup feeding, bottle feeding, feeding of solid foods, the bath, play, sleep, the social attitude of the child, his activity on the floor and other activities. The two volumes constitute a complete record of the usual activities of the infant under normal conditions and should prove invaluable not only to one working along this line, but to any physician who wishes to use such material as a basis for estimating behavior conditions in infants.

**Die Armlähmungen bei Neugeborenen.** By Geh. Med.-Rat. Prof. Dr. Erwin Kehrer. Monograph reprinted from Zeitschrift für Geburtshilfe und Gynäkologie, Band 107. Price, 10.40 marks. Pp. 90, with 20 illustrations. Stuttgart: Ferdinand Enke, 1934.

Kehrer's monograph on injuries to the arm in new-born infants as a result of trauma at birth is an excellent reference book for the pediatrician and obstetrician. In it is clearly presented the anatomic and physiologic background of these conditions, knowledge of which is essential for accuracy in exact localization of lesions by physical examination. A drawing shows dissection of the nerves of the neck and arm which are especially vulnerable to trauma at birth; a chart shows each nerve, the muscle it innervates and the function of that muscle. Copies of these might well be in every pediatrician's notebook.

The author discusses five types of injuries to the arm, including the classic Duchenne-Erb type, the Klumpke type and a combination of these, as well as an atypical, partial injury of more than one nerve, which he terms a "rudimentary arm-injury," and, lastly, a "combination" type, or injury to a nerve to a muscle of the arm combined with injury to some other nerve in close proximity, such as the facial, hypoglossal or accessory nerves. A discussion is then presented of intracranial hemorrhage due to injury at birth resulting in damage to that area in the brain which controls the nerves to the arm. Lastly, the author takes up spinal injuries which may occur when plexuses and roots are injured as a result of traction.

The differential diagnosis between the different types of peripheral lesions and between peripheral and central lesions is ably discussed and is illustrated by photo-

graphs of infants and by histories of cases taken from the literature as well as from the author's own material. The importance is pointed out of differentiating all these injuries from "pseudo-arm plexus injuries," such as syphilis (Parrot's pseudoparalysis), osteomyelitis, fractures, and other lesions.

The monograph is completed with a discussion of the prognosis, prophylaxis and therapy. A comprehensive bibliography is included. This monograph on a subject of great importance, which is rather briefly discussed in most textbooks, is of such value that it might well be translated into English for more extensive use in this country.

**Textbook of Bacteriology.** By Zinsser and Bayne-Jones. Seventh edition. Price, $8. Pp. 1,226, with 174 illustrations. New York: D. Appleton-Century Company, 1934.

This is a book which will delight both the bacteriologist and the practitioner of medicine with its well balanced array of material. This seventh edition by Zinsser and Bayne-Jones has developed into a manual of infectious diseases as well as a text on bacteriology.

The advances in bacteriology during the past several years have been of great importance, and revisions of older concepts are necessary. The greatest commendation of this book is that it is capably written and is thoroughly up to date. Revisions were made even during publication, so that the latest discoveries are embodied.

The first section of the book deals with the general biology of bacteria, including bacterial variation and dissociation. The section "Infection and Immunity" is of special interest because of the advances in immunology which have been due partly to new lines of thought predicated on the correlation of bacterial change and virulence.

The third and largest section, of course, describes the pathogenic microorganisms and their relation to disease. In addition there are six other sections: "The Rickettsia Diseases," "The Spirochetes," "Medical Mycology," "Viruses," "Outline of Diagnostic Methods of Medical Protozoology" and "Technical Methods of Bacteriology, Immunology and Serology."

The rapidly growing knowledge of ultramicroscopic agents has greatly increased the importance of viruses.

In this edition the chapters on the bacteriology of water, air and soil have been eliminated to allow space for newer material incorporated in the rest of the book.

**Diagnostik und Therapie der Kinderkrankheiten.** By Dr. F. Lust. Eighth Edition. Pp. 573. Price, 11.50 marks. Berlin: Urban & Schwarzenberg, 1934.

The eighth edition of this German textbook of pediatrics has been entirely revised. All the common disorders of infants and children and many of the rare diseases are discussed from the standpoint of diagnosis and treatment. Under diagnosis the essential symptoms, physical findings and laboratory procedures are concisely described. Therapy is considered in much more detail and is given much more space than diagnosis.

There is one chapter on therapeutic technic, in which are described such procedures as blood transfusion, sinus puncture and intubation.

The division on materia medica is complete. The physical and chemical properties of drugs are briefly described, and the recommended dose of each drug for infants and children of various ages is suggested. The last chapter gives a list of the spas and health resorts suitable for sick children, with a discussion of the chief advantages of each.

# Obituary

## PAUL GALPIN SHIPLEY, M.D.
1888–1934

Dr. Paul Galpin Shipley was born in 1888 and graduated from Yale University in 1909 and from the medical school in 1913. While a student in the medical school, he worked in the department of zoology under Professor Ross Harrison and won a friendship and esteem from Dr. Harrison which lasted to the time of Dr. Shipley's death. After graduation from Yale, through the influence of Dr. Harrison, he entered the department of Dr. Franklin P. Mall at Johns Hopkins University, where he remained four years. During this time he mastered the technic of histologic research and became an authority on mitochondria and the reaction of tissue cells to the vital benzidine dyes. In his subsequent investigations in the field of pediatrics his knowledge of anatomy was an enormous asset and determined the course of his investigations.

In 1917, impelled by a great natural interest in human beings, which was a salient characteristic throughout his life, he joined the department of pediatrics at Johns Hopkins University School of Medicine under Dr. Howland, in which he later became associate professor, and immediately embarked on studies of great importance. These studies, which were carried on in conjunction with McCollum, Simmonds and Park, yielded fundamental information on the cause and nature of rickets as the result of the successful production of the disease in the rat. Among other discoveries, he found, with the investigators already mentioned, that starvation initiates a healing process in rickets and produced the first experimental proof that cod liver oil causes deposition of lime salts in rachitic cartilage and bone. With these investigators he also showed that rickets could be produced through the addition to the diet of strontium, magnesium and barium. With Dr. McCollum he proved the separate existence of the antirachitic vitamin. Dr. Shipley was the first to produce calcification of living cartilage in vitro, and this observation made possible the subsequent important studies by Holt and La Mer and Kramer, Shelling and Orent on the factors involved in calcification of cartilage and bone. Outside the field of rickets some of Shipley's studies were of great importance. With Blumberg and Scott he worked out a method for the detection of lead in the blood by means of the spectroscope. This method for the detection of lead poisoning exceeds all others in delicacy and accuracy. One of his most recent studies was on tick fever in children, and his paper on that subject in the *Bulletin of the Johns Hopkins Hospital* (**51:** 83, 1932) is a classic presentation. His intellectual interests and the subjects of

his writings were extremely varied and included medical history. His paper on the treatment of convulsions with calcium in the seventeenth century and on "Our Fathers and the Scorby" are interesting and entertaining historical presentations. In addition to his many scientific papers, he wrote numerous valuable articles for textbooks.

As an investigator Dr. Shipley stood in the front rank and was one of the internationally known figures in American pediatrics. By nature he was a scholar and could speak authoritatively on a variety of subjects unfamiliar to the ordinary mind. Heraldry and the history of firearms and of archery were subjects on which he could have written with ease. In conversation he showed astonishing information on mythology, medieval history and almost any subject which happened to come up for discussion. He possessed a capacious mind which seemed to be able to hold permanently all the facts which had entered. His knowledge was readily available to himself, so that he wrote with ease and fluency.

Dr. Shipley was a most kindly man and had hosts of friends both in this country and abroad. He was a delightful companion and was universally beloved. As a clinician he excelled particularly in diagnosis and was an authority in the Harriet Lane clinic on a variety of obscure conditions. During a short journey to Central America in 1927 he obtained a remarkable knowledge of tropical diseases affecting the child.

Dr. Shipley died suddenly of coronary thrombosis on Sept. 12, 1934. In his death pediatrics in this country has lost one of its most brilliant minds, one of its foremost scholars and investigators and a most delightful character and man.

E. A. P.

# Directory of Pediatric Societies *

## INTERNATIONAL

### International Association of Preventive Pediatrics

President: Professor G. Scheltema, 6a Heereplein, Groningen, The Netherlands.
Secretary: Dr. Daniel Oltramare, 15 Rue Lévrier, Geneva, Switzerland.
Place: Bâle, Switzerland: Time: September, 1935.

## FOREIGN

### British Paediatric Association

President: Dr. Hugh Thursfield, 84 Wimpole St., London, W. 1.
Secretary: Dr. Arthur G. Maitland Jones, 31 A Weymouth St., London, W 1.
Place: Newcastle, County Down, Ireland. Time: May 3-4, 1935.

### Royal Society of Medicine, Section for the Study of Disease in Children

President: R. C. Jewesbury, Esq., M.D., 5 Wimpole St., W. 1, London.
Secretary: Dr. Helen Mackay, 28 John St., W. C. 1, London.
Place: 1 Wimpole St., London. Time: Fourth Friday of each month, 4:30 p. m.

## NATIONAL

### American Medical Association, Scientific Assembly, Section on Diseases of Children

Chairman: Dr. A. Graeme Mitchell, Children's Hospital, Cincinnati.
Secretary: Dr. Ralph M. Tyson, 334 S. 21st St., Philadelphia.
Place: Atlantic City, N. J. Time: June 10-14, 1935.

### American Academy of Pediatrics

President: Dr. Thomas B. Cooley, 1728 Seminole Ave., Detroit.
Secretary: Dr. C. G. Grulee, 636 Church St., Evanston, Ill.
Place: New York. Time: June 7-8, 1935.
Region I. Place: Philadelphia. Time: Fall, 1935.
Regions II and III. Place: St. Louis. Time: Fall, 1935.
Region IV. Place: Seattle. Time: Fall, 1935.

### American Child Health Association

President: Dr. Samuel McC. Hamill, 1822 Spruce St., Philadelphia.
Secretary: Dr. Philip Van Ingen, 125 E. 71st., New York.

### American Hospital Association, Children's Hospital Section

President: Mr. Robert Witham, Children's Hospital, Denver.
Secretary: Miss Agnes O'Roke, Kosair Crippled Children's Hospital, Louisville, Ky.

### American Pediatric Society

President: Dr. Borden S. Veeder, 3720 Washington Ave., St. Louis.
Secretary-Treasurer: Dr. Hugh McCulloch, 325 N. Euclid St., St. Louis.
Place: Wade Park Manor, Cleveland. Time: May 2-4, 1935.

### Canadian Society for the Study of Diseases of Children

President: Dr. D. P. Arnold, 159 Bidwell Parkway, Buffalo, N. Y.
Secretary-Treasurer: Dr. R. R. Struthers, 1414 Drummond St., Montreal.
Place: Buffalo, N. Y. Time: June 1935.

### Society of Pediatric Research

President: Dr. Joseph Stokes Jr., Children's Hospital, Philadelphia.
Secretary: Dr. A. A. Weech, Babies Hospital, New York.
Place: Atlantic City, N. J. Time: May 1935.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

## SECTIONAL

### North Pacific Pediatric Society

President: Dr. Armin Rembe, Stimson Bldg., Seattle.
Secretary: Dr. Frank H. Douglass, 1227 Medical and Dental Bldg., Seattle.

### Southern Medical Association, Section on Pediatrics

Chairman: Dr. Oliver W. Hill, 301 Medical Arts Bldg., Knoxville, Tenn.
Secretary: Dr. Madison Hines Roberts, 104 Ponce de Leon Ave., N. E., Atlanta, Ga.

## STATE

### Alabama Pediatric Society

President: Dr. Charles Abbott, 1817 Broad St., Tusaloosa.
Secretary-Treasurer: Dr. Wallace Clyde, Employee's Hospital, Fairfield.
Place: Mobile. Time: April 17-19, 1935.

### Georgia Pediatric Society

President: Dr. Benjamin Bashinski, 700 Spring St., Macon.
Secretary-Treasurer: Dr. Roger W. Dickson, 33 Ponce de Leon Ave., N. E., Atlanta.

### Indiana State Pediatric Society

President: Dr. Louis H. Segar, 23 E. Ohio St., Indianapolis.
Secretary-Treasurer: Dr. Matthew Winters, 23 E. Ohio St., Indianapolis.
Time: Two meetings a year.

### Louisiana State Pediatric Society

President: Dr. John Signorelli, 2000 Tulane Ave., New Orleans.
Secretary-Treasurer: Dr. Edwin A. Socola, 1027 Canal Bank Bldg., New Orleans.
Place: New Orleans. Time: April 29, 1935.

### Medical Society of State of New York, Section on Pediatrics

Chairman: Dr. George Sincerbeaux, 6 William St., Auburn.
Secretary: Dr. John D. Craig, 12 E. 11th St., New York.

### Michigan State Medical Society, Pediatric Section

Chairman: Dr. Edgar E. Martmer, 749 David Whitney Bldg., Detroit.
Secretary: Dr. C. R. Dengler, 131 W. Michigan Ave., Jackson.
Place: Flint. Time: September 1935.

### Mississippi State Pediatric Society

President: Dr. N. C. Womack, Jackson Infirmary, Jackson.
Secretary-Treasurer: Dr. Guy C. Jarratt, 920 Crawford St., Vicksburg.
Place: To be designated by Society. Time: Yearly.

### Nebraska Pediatric Society

President: Dr. J. A. Henske, 1614 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. J. H. Murphy, 915 Medical Arts Bldg., Omaha.
Place: As announced by committee. Time: Third Thursday of each month from October to June, inclusive. Dinner at 6 p. m.

### North Carolina Pediatric Society

President: Dr. J. Buren Sidbury, 15 N. Fifth St., Wilmington.
Secretary: Dr. E. K. McLean, 121 W. Seventh St., Charlotte.
Place: Various children's hospitals. Time: Twice a year.

### Oklahoma State Pediatric Society

President: Dr. C. V. Rice, 222 Wall St., Muskogee.
Secretary: Dr. B. H. Nicholson, 301 N. W. Twelfth St., Oklahoma City.
Place: Oklahoma City. Time: May 1935.

### Pennsylvania Medical Society, Pediatric Section

Chairman: Dr. Francis T. O'Donnell, 345 North Main St., Wilkes-Barre.
Secretary: Dr. Theodore O. Elterich, 119 S. Highland Ave., Pittsburgh.
Place: Harrisburg. Time: Sept. 30-Oct. 3, 1935.

### South Carolina Pediatric Society

President: Dr. Julian P. Price, Medical Bldg., Florence.
Secretary-Treasurer: Dr. Leland B. Salters, 317 S. Warley Ave., Florence.
Place: Columbia. Time: January 1935.

### Tennessee State Pediatric Society

President: Dr. Arthur G. Jacobs, 1193 Madison Ave., Memphis.
Secretary: Dr. Joe T. Smith, 702 Medical Arts Bldg., Knoxville.
Place: Nashville. Time: April 1935.

### Texas Pediatric Society

President: Dr. Ramsay H. Moore, 3600 Fairmount St., Dallas.
Secretary-Treasurer: Dr. F. H. Lancaster, Medical Arts Bldg., Houston.

### Virginia Pediatric Society

President: Dr. W. P. McDowell, 142 W. York St., Norfolk.
Secretary: Dr. W. A. McGee, 616 W. Grace St., Richmond.

### West Virginia State Medical Society, Section on Pediatrics

President: Dr. R. C. Hood, 428 W. Pike St., Clarksburg.
Secretary: Dr. Russell C. Bond, 1325 Chapline St., Wheeling.

## LOCAL

### Academy of Medicine of Brooklyn, Pediatric Section

President: Dr. Harry M. Greenwald, 499 Ocean Ave., Brooklyn.
Secretary: Dr. Sylvan D. Lazarus, 847 Lincoln Place, Brooklyn.
Place: Medical Society of County of Kings Bldg., 1313 Bedford Ave., Brooklyn.
Time: Third Wednesday of each month from October to May, 8:30 p. m.

### Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. J. A. Garvin, Carnegie Medical Bldg., Cleveland.
Secretary: Dr. William M. Champion, 2323 Prospect Ave., Cleveland.
Place: Cleveland Medical Library Bldg., Time: October, December, February and April.

### Academy of Medicine, Toronto, Section of Pediatrics

President: Dr. C. V. Mulligan, 17 Walmer Rd., N., Toronto.
Secretary: Dr. T. B. Verner, 170 St. George St., Toronto.

### Bronx Pediatric Society

President: Dr. Morris Gleich, 9 Post Ave., New York.
Secretary: Dr. Harry D. Pasachoff, 1560 Grand Concourse, New York.
Place: Concourse Plaza Hotel, 161st St. and Grand Concourse. Time: Every second Wednesday of each month, except June, July, August and September.

### Brooklyn Academy of Pediatrics

President: Dr. Carl H. Laws, 12 Pierrepont St., Brooklyn.
Secretary: John A. Monfort, 861 Carroll St., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Fourth Wednesday of each month, except May, June, July, August and September.

### Central New York Pediatric Club

President: Dr. Frank Vander Bogert, 111 Union St., Schenectady.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Place: Various cities in New York. Time: Third Thursday of April and September.

### Chicago Pediatric Society

President: Dr. Stanley Gibson, 9503 Prospect Ave., Chicago.
Secretary: Dr. James H. Wallace, 715 Lake St., Oak Park, Ill.
Place: John B. Murphy Memorial Bldg. Time: Third Tuesday of each month.

### Dallas County Pediatric Society

President: Dr. P. E. Luecke, 4105 Live Oak St., Dallas, Texas.
Secretary: Dr. R. S. Usry, 1835 Garrett Ave., Dallas, Texas.
Place: Bradford Memorial Hospital. Time: 1 p. m., first and third Saturdays of each month.

### Detroit Pediatric Society

President: Dr. Earl W. May, 1551 Woodward Ave., Detroit.
Secretary: Dr. E. A. Wishropp, 10 Peterboro Ave., Detroit.
Place: Wayne County Medical Society. Time: 8:30 p. m., first Wednesday of each month.

### Fulton County Medical Society, Pediatric Section (Atlanta, Ga.)

Chairman: Dr. Joseph Yampolsky, 478 Peachtree St., Atlanta.
Secretary: Dr. T. I. Willingham, 384 Peachtree St., Atlanta.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: Second Thursday of each month from October to April, 8 p. m.

### Houston Pediatric Society

President: Dr. Byron P. York, 3820 Fannin St., Houston, Texas.
Secretary-Treasurer: Dr. Edward O. Fitch, 3717 Main St., Houston, Texas.
Place: Houston Club: Time: Third Monday of each month.

### Intermountain Pediatric Society

President: Dr. Helmina Jeidell, 11 Exchange Pl., Salt Lake City, Utah.
Secretary-Treasurer: Dr. Edwin R. Murphy, 9 Exchange Pl., Salt Lake City, Utah.
Place: Salt Lake City, General Hospital. Time: First Thursday of each month, 8 p. m.

### Kansas City (Missouri) Pediatric Society

President: Dr. Harry M. Gilkey, 1103 Grand Ave., Kansas City.
Secretary: Dr. S. A. Stadler, 6133 Brookside Blvd., Kansas City.
Place: Kansas City General Hospital. Time: On call.

### Medical Society of the County of Queens, Inc., Section on Pediatrics

Chairman: Dr. Henry A. Reisman, 148, 88th Ave., Jamaica, L. I.
Secretary-Treasurer: Dr. Edith A. Mittell, 112 Queens Blvd., Forest Hills, N. Y.
Place: Queens County Medical Bldg., Forest Hills, N. Y. Time: Third Monday of October, January, March and May.

### Memphis Pediatric Society

President: Dr. Harry J. Jacobson, 1193 Madison Ave., Memphis, Tenn.
Secretary-Treasurer: Dr. Clyde V. Croswell, 1052 Madison Ave., Memphis, Tenn.
Place: Memphis General Hospital. Time: Second Tuesday of each month, except June, July and August, 8 p. m.

### Milwaukee Pediatric Society

President: Dr. F. R. Janney, 1411 Wauwatosa Ave., Wauwatosa, Wis.
Secretary: Dr. John H. Reynolds, 1628 Wisconsin Ave., Milwaukee.
Place: Children's Hospital. Time: Second Wednesday of each alternate month, beginning with February.

### New England Pediatric Society

President: Dr. Paul Emerson, 319 Longwood Ave., Boston.
Secretary-Treasurer: Dr. Henry E. Gallup, 66 Commonwealth Ave., Boston.
Place: Boston Medical Library. Time: Four meetings a year, occurring from September to May.

NEW YORK ACADEMY OF MEDICINE, SECTION OF PEDIATRICS

Chairman: Dr. Harry Bakwin, 132 E. 71st St., New York.
Secretary: Dr. Alexander T. Martin, 107 E. 85th St., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

NORTHWESTERN PEDIATRIC SOCIETY

President: Dr. O. W. Rowe, 205 W. 2nd St., Duluth, Minn.
Secretary: Dr. E. F. Robb, 1801 Lyndale Ave., S. Minneapolis.
Place: Minneapolis. Time: Spring, 1935.

PHILADELPHIA PEDIATRIC SOCIETY

President: Dr. John D. Donnelly, 115 Bryn Mawr Ave., Bala, Pa.
Secretary: Dr. James E. Bowman, 4091 Comly St., Philadelphia.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday of each month from October to June, inclusive.

PITTSBURGH PEDIATRIC SOCIETY

President: Dr. Carl K. Wagener, Jenkins Arcade, Pittsburgh.
Secretary-Treasurer: Dr. E. R. McClusky, 3200 Fifth Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Tuesday, alternate months from October to April, inclusive.

RICHMOND PEDIATRIC SOCIETY

President: Dr. S. A. Anderson Jr., 2326 W. Grace St., Richmond, Va.
Secretary-Treasurer: Dr. Emily Gardner, 1100 W. Franklin St., Richmond, Va.
Place: Richmond Academy of Medicine, 1200 E. Clay St. Time: First Thursday of each month (except July, August and September).

ROCHESTER PEDIATRIC SOCIETY

President: Dr. H. F. Rowley, 176 S. Goodman St., Rochester, N. Y.
Secretary: Dr. J. M. Parker, 26 S. Goodman St., Rochester, N. Y.
Place: Arranged by program committee. Time: Second Friday of each month.

ROCKY MOUNTAIN PEDIATRIC SOCIETY

President: Dr. Arthur L. Esserman, Republic Bldg., Denver.
Secretary: Dr. Ralph Verploeg, 19th and Gilpin Sts., Denver.
Place: Children's Hospital, Denver. Time: First Saturday of each month from September to May, inclusive, 2:30 p. m.

ST. LOUIS PEDIATRIC SOCIETY

President: Dr. J. V. Cooke, St. Louis Children's Hospital, St. Louis.
Secretary-Treasurer: Dr. Katherine Bain, Beaumont Bldg., St. Louis.
Place: St. Louis Medical Society. Time: Third Friday of every month.

SEATTLE PEDIATRIC SOCIETY

President: Dr. Armin C. Rembe, Stimson Bldg., Seattle.
Secretary: Dr. Frank H. Douglass, Medical-Dental Bldg., Seattle.
Place: College Club. Time: Third Friday of each month, 6:30 p. m.

SOUTHWESTERN PEDIATRIC SOCIETY

President: Dr. Victor E. Stork, 1136 W. Sixth St., Los Angeles.
Secretary: Dr. Howard R. Cooder, 3875 Wilshire Blvd., Los Angeles.
Place: University Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

UNIVERSITY OF MICHIGAN PEDIATRIC AND INFECTIOUS DISEASE SOCIETY

President: Dr. Daniel Budson, 3001 W. Grand Blvd., Detroit.
Secretary: Dr. D. Murray Cowie, University of Michigan, Ann Arbor, Mich.
Place: Ann Arbor, Mich. Time: Nov. 22-23, 1935.

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